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# SECTION ON

# OPHTHALMOLOGY

OF THE

# AMERICAN MEDICAL ASSOCIATION

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LOS ANGELES, JUNE 27-30, 1911

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# TO THE MEMBERS OF THE SECTION ON OPHTHALMOLOGY

OF THE

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All the completed papers for the program of the Section on Ophthalmology of the American Medical Association, Los Angeles Session, 1911, will be found herein, and, in most cases, in the order in which they will be presented for discussion. Provision has been made for notes on the margins of the pages and on the blank page following each paper.

Following the plan in force during the last few years, the papers will not be read before the Section but each author will be allowed not to exceed ten minutes in which to summarize the points in his paper and to introduce discussion. The member appointed to open the discussion of any paper will be allowed not to exceed ten minutes and subsequent speakers five minutes. Any one who has written out his discussion must of course hand it to the stenographer, if it is to be used.

Attention is called to the desire on the part of the officers of the Section to have an exhibition of pathologic specimens, photographs, charts and printed histories of rare and interesting cases. Members are also invited to exhibit new instruments and appliances which may be of interest to the members of the Section. Those desiring to contribute to this feature are requested to correspond with the secretary of the Section.

The officers and executive committee desire to thank the essayists for their cooperation in making it possible to issue the presession volume on time.

EDGAR S. THOMSON, Secretary.
ALBERT E. BULSON, JR., Chairman.

# THE BOUND TRANSACTIONS

All of the within papers, together with the formal discussion of the same at the Los Angeles session, will be printed and bound in cloth, thus constituting the Transactions of the Section of Ophthalmology of the American Medical Association for 1911. Members of the Section and others may obtain these bound Transactions at $1 a volume by sending subscriptions to the American Medical Association, 535 Dearborn Avenue, Chicago, on or before July 15, 1911. After the volume is published the price will be $1.50.

Edgar S. Thomson, Secretary.

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# THE HEALING OF IRIDECTOMY WOUNDS AND ITS INFLUENCE ON GLAUCOMA *

ADOLF ALT, M.D.
ST. LOUIS

Glaucoma in its different forms, being as yet but partially understood, must needs still arouse the interest and particular study of every student. It is not astonishing, therefore, that the literature on the subject has reached an enormous magnitude and that the theories evolved concerning the etiology as well as the cure of this many-sided disease are becoming increasingly numerous.

It is not the object of this paper to go over the whole question; I intend to confine myself to the question of the healing of the iris wounds after iridectomy and to see in what manner the iridectomy may exert a curative influence in the course of a glaucoma.

In 1874[1] I made a large number of experimental iridectomies on rabbits in order to study the manner in which iridectomy wounds heal. I was aware then and am now that we cannot unhesitatingly apply experimental results obtained on animals' eyes to the human eye. Yet, in this particular case and in general in the healing of wounds, my histologic studies have shown no marked difference between the eye of a rabbit and that of man.

The normal healing of the wound in an iris which was healthy and which was not infected during the operation, according to my experiments, gave results as follows:

After the fibrinous coagulum containing erythrocytes, which in the first few days covers the iris wound, has been cast off or absorbed, the stump of the iris, in the microscopic section, appears in most cases swollen and

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

[1] Anatomic Changes Following Iridectomy, Arch. Ophth., iv, 423.

knob-like, or at least rounded off; in others the wound edge is slanting obliquely with the more or less sharply pointed margin forward toward the anterior chamber or backward toward the posterior chamber.

The rounded-off or knob-like end of the stump after a few days, even, no longer presents a wounded surface. Its posterior part is covered with pigment epithelium, its anterior one with endothelial cells. Sometimes the cells of one of the two surfaces predominate. Where the cells of both iris surfaces meet on the stump, they are often bent hook-like into the former wound, apparently drawn inward into the stroma of the iris by the retraction of its tissue. The cellular proliferation and the infiltration which follow the operation disappear in a few days.

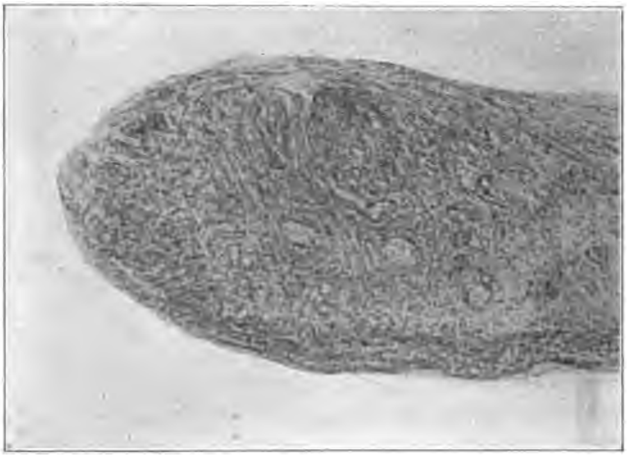

Fig. 1.—The stump of the iris from a white rabbit four days after the operation healed *per primam*. Stained with carmin.

When the iris has been cut obliquely, the sharp-angled flap is gradually bent back toward the obtuse one and thus the wound is closed after from six to eight days, and the stump is covered either with endothelium or with pigment epithelium according to the side the flap was on.

The first manner of healing of the iris stump may be called healing *per primam intentionem,* the second manner *per secundam intentionem.*

It appeared clear that the manner of healing depended on the direction of the cut made by the scissors, which, in the former produced two more or less equal flaps, which promptly collapsing closed the gap, while in the other case only one, either an anterior or a posterior flap, resulted, which by the contraction of the iris tissue was gradually bent over either backward or forward and thus the wound surface became closed. Figures 1 and 2 are from photographs of some specimens made at the time of these experiments.

Among the many microscopic sections made of these series of rabbits' eyes on which I had performed an iridectomy, when no complications interfered, the healing and covering over of the wound surface invariably took place in from a few hours to about eight days after the operation had been performed.

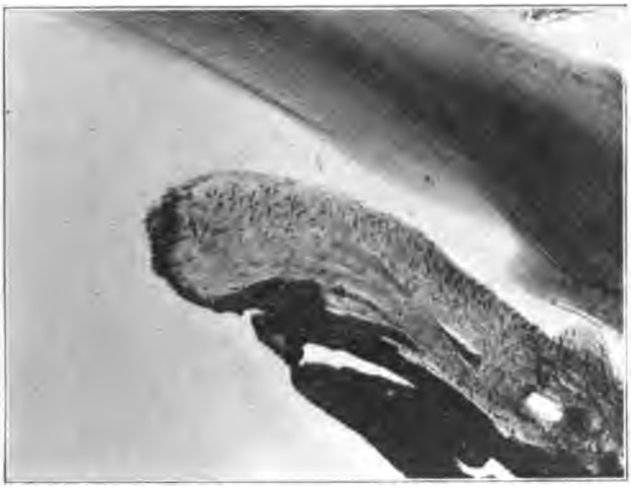

Fig. 2.—The stump of the iris from a dark rabbit eight days after operation, also healed *per primam*.

Through the kindness of the late Prof. Otto Becker. I was given an opportunity to examine a number of specimens from human eyes on which an iridectomy had been performed and, comparing these with my rabbits' specimens, we came to the conclusion that the manner of healing was very much the same in man as in the rabbit.

Ever since, I have carefully examined the few human eyes that I could obtain on which an iridectomy had been performed. Their number is not very large, and except in eyes on which a combined cataract extraction had been performed, the iris was probably diseased in every one before the iridectomy was made. The eyes from which a cataract had been extracted were enucleated on account of panophthalmitis and, therefore, also yielded no sections with a healthy iris. However, when the iris stump was not entangled in the wound or in an older cicatrix the conditions were not unlike those described above in the rabbit.

Yet I have seen a few iris stumps in which the wound did not seem to have healed in exactly the same manner.

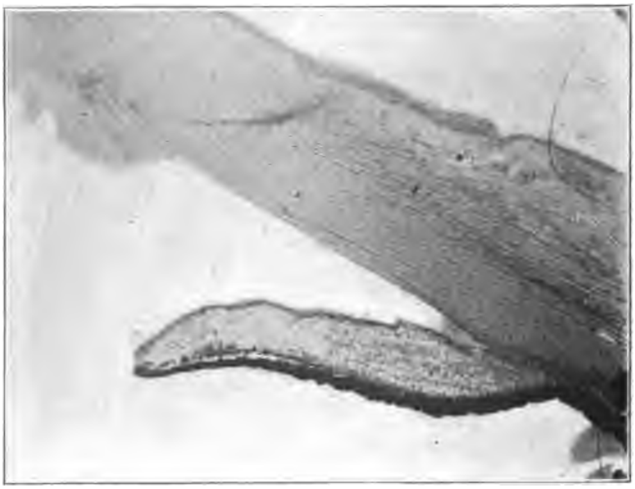

Fig. 3.—The wound of the iris stump is closing over, although the process does not seem quite accomplished. The pigment epithelium is slightly loosened from the iris tissue. This is probably a post-mortem occurrence. Primary union does not seem to have been quite accomplished in this case.

In these sections, it was either prevented from closing in the manner described by a blood-clot or fibrinous material at the time I examined it, or, in a number of eyes which had been operated on a long time previously, it was possible to demonstrate after all a thin layer made up of flat spindle cells coming from the anterior endothelium covered the apparently unclosed wound. There was in reality no raw surface.

The conditions of the wounds on the pillars of the coloboma were the same as at the base of the coloboma.

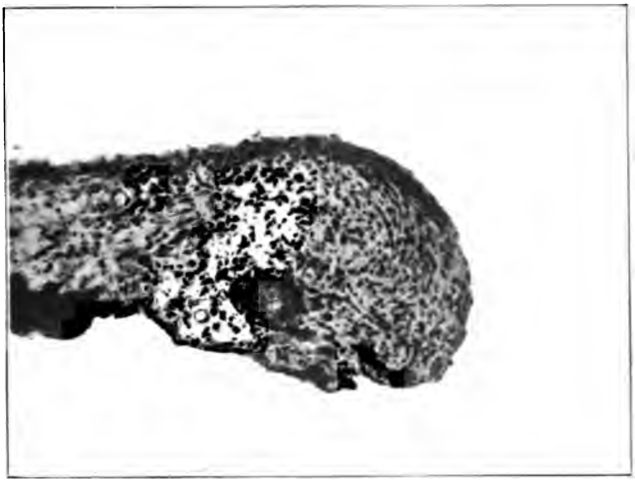

Figure 4

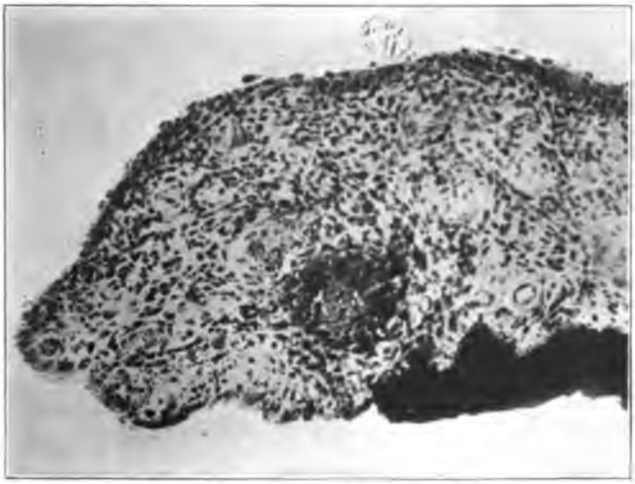

Figure 5

Figs. 4 and 5.—These illustrations give excellent examples of the primary union from an eye which was enucleated eight days after an iridectomy had been performed. In both of these figures there were apparently two flaps, an anterior one and a posterior one, the anterior one being the thicker, at least in Figure 5. The flaps have almost healed together and the line of contact can still be recognized by several rows of cells. The endothelium of the anterior surface seems to be creeping over the small gap. The pigment epithelium has for some distance fallen off, a condition which may be due to handling post mortem. The tissues are considerably infiltrated with round cells.

In a paper entitled, "A Histologic Study of the Normal Healing of Wounds after Cataract Extraction," Thomas Henderson[2] in his Figure 2, draws such a rounded-off iris stump, but does not refer to it in the text. From this drawing, it seems that the iris wound made during a combined extraction was healed, or healing in the manner here described, when the eye was removed twelve days after extraction. The paper is written to show that the iridectomy wounds resulting from a combined cataract extraction on a normal iris never heal.

In a paper entitled, "The Histology of Iridectomy,"[3] the same author comes to the conclusion that "the severed iris tissue never shows any sign of scar, or other manifestation of reaction, for even years after the cut surface appears as if the operation had been performed post mortem."

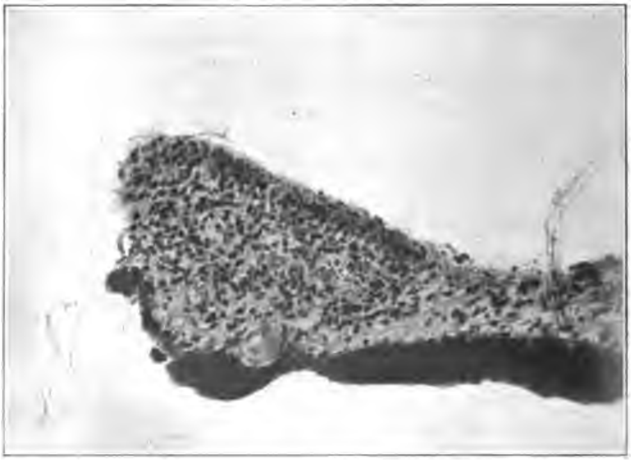

Fig. 6.—The stump is several weeks old and, although peculiar in its rather square than rounded-off outline, it seems to have healed by first intention. The posterior flap is covered by an interrupted pigment layer (post mortem); the anterior flap, also, does not seem to possess a continuous endothelial covering, yet there is no bare, raw surface.

In a recent book on glaucoma, Henderson[4] still further elaborates this same view and says: "The iris tissue forming the base and pillars of the coloboma always remains an open, raw, unhealed area, in free and open communication with the aqueous of the anterior chamber, in exactly the same manner as the crypt surfaces." The drawings in this chapter are repeated from the previous papers and refer to eyes on which a combined cataract extraction had been performed. Henderson bases his views on the effect of an iridectomy in glaucoma on the fact that iris wounds never heal.

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[2] Ophth. Rev., May, 1907.
[3] Ophth. Rev., July, 1907.
[4] Henderson: Glaucoma, London, 1910, E. Arnold, p. 187.

These results of Henderson's researches prompted me to study again all the specimens from human eyes in my collection which might have some bearing on this interesting question, the more so since, if Henderson's observations were correct, the effect of an iridectomy on the course of glaucoma could be more easily understood.

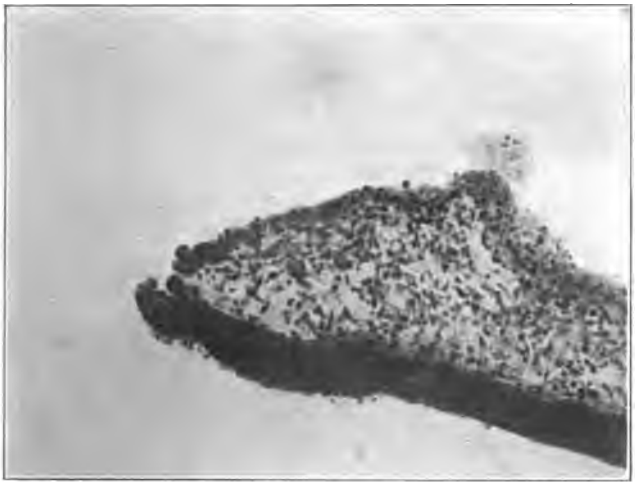

Fig. 7.—The stump has been cut off very obliquely. The larger part of the wound is covered with endothelium; the pigment layer reaches forward over the sharp angle to a small distance to where the two layers meet. The flap has evidently not been bent over the wound surface, as in other cases.

I was rather astonished to find that in almost all my human specimens of iridectomy stumps the iris tissue showed decided signs of inflammation; yet, since all came from eyes which it had been necessary to enucleate, this was, after all, quite natural, even in the sections from an eye which, after having been operated on previously by iridectomy, had been removed on account of a chorioidal sarcoma.

The illustrations in Figures 3 to 18 reproduce some of the characteristic sections:

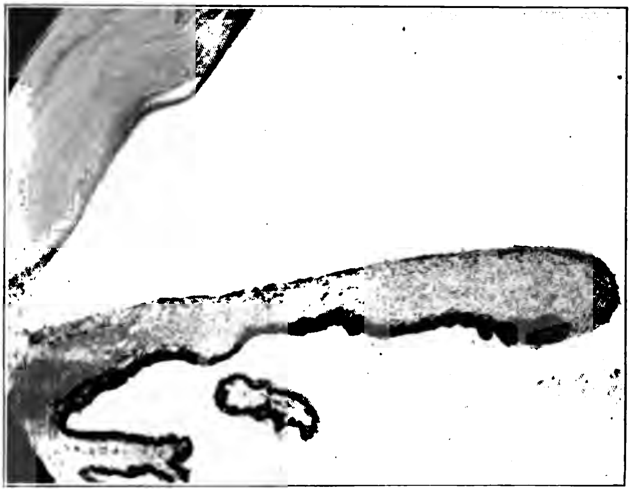

Figure 8

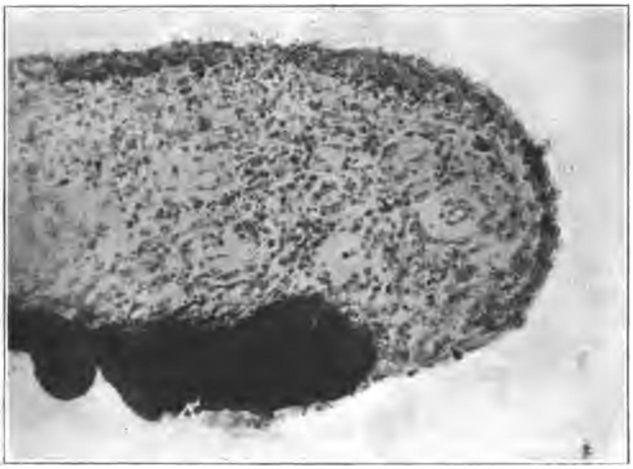

Figure 9

Figs. 8 and 9.—There seems to have been a long anterior flap which has, so to speak, fallen backward toward the pigment epithelium and thus closed the gap of the wound. The resulting round stump is perfectly covered over by anterior endothelium.

Similar pictures from glaucomatous eyes with a healed stump are given by Henderson, who in this connection states that "when a stimulus is supplied by toxic or other agents cicatrization and sclerosis of the iris tissue follows in the same manner and governed by the same principles as elsewhere in the body."

According to this author, therefore, iridectomy wounds or other wounds made in the iris tissue when this is in a normal condition are never closed; such a closure takes place only when the iris has been previously diseased, as shown by atrophy and sclerosis, or becomes so, for instance, by infection during the operation. That is, according to his views, the healthy tissue of the iris, although provided with numerous blood-vessels in an active and functioning state, shows no reaction after being wounded, while the sclerosed and atrophied iris, as found in chronic glaucoma, seals up its wounds.

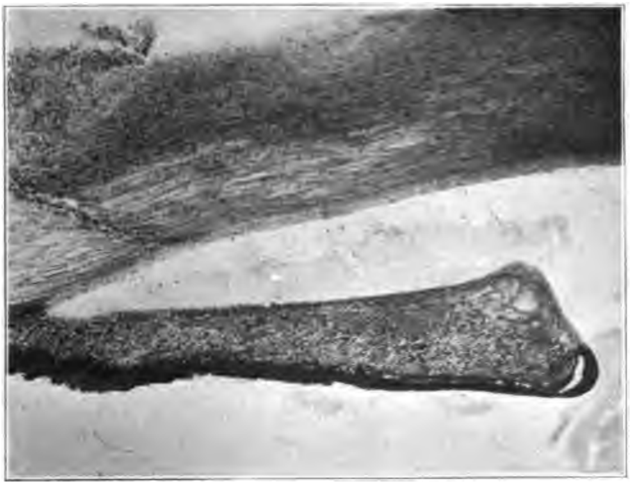

Figure 10

In 1896[5] I had occasion to describe an eye with iridodialysis and cyclodialysis in which I said:

> The detached iris tissue, besides being considerably contracted toward the sphincter edge, is atrophic, and contains a large quantity of blood-pigment and of uveal pigment, which is partly enclosed in cells, partly free. The walls of its blood-vessels are considerably thickened. The uveal layer is very thick, wrinkled and folded backward. The sections do not show any union of what may be termed the anterior and pos-

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[5] Am. Jour. Ophth., October, 1896, p. 296.

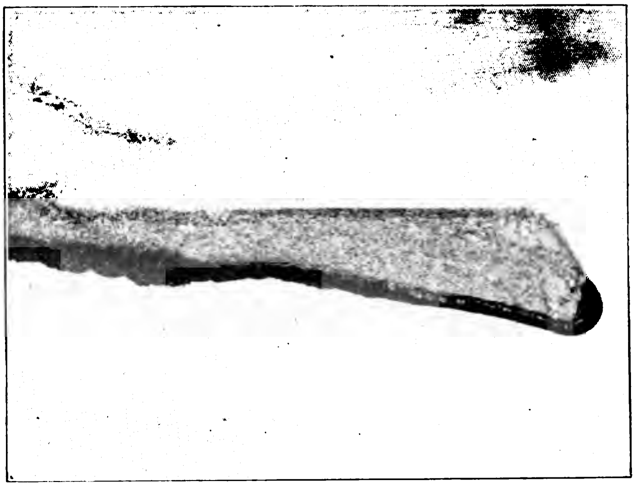

Figure 11

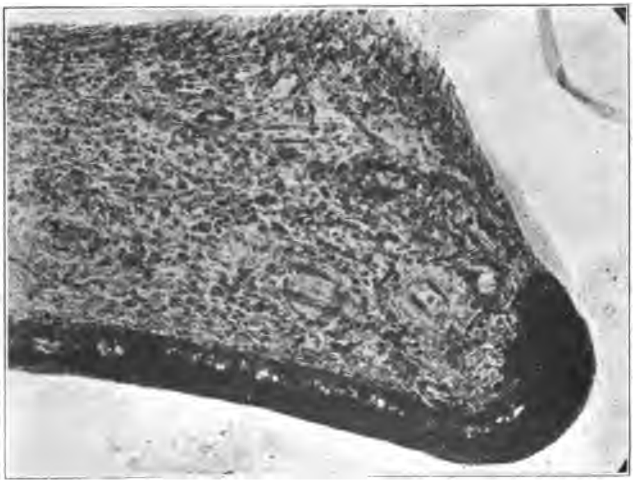

Figure 12

Figs. 10, 11 and 12.—The posterior flap seems to have been the longer one. The wound surface is perfectly covered over, the pigment epithelium reaching forward to quite a distance on the stump. A newly formed endothelial membrane covers the anterior surface of the whole iris, as well as the stump. This membrane, which is never wanting in older cases and is present in most quite recent cases of glaucoma, and which is the cause of the ectropium uveæ, which is also found as a rule in glaucomatous eyes, in these specimens seems to have pulled the pigment epithelium forward. The peculiar loosening of the pigment epithelium layer from its contact with the posterior surface of the iris may be the consequence of such a pull; it may also be that this patient suffered from diabetes, in which such a condition of the pigment epithelium seems the rule.

**terior flap of the tear, as I have seen it many times after an iridectomy or injury to the iris, etc. In this specimen there is simply a ragged edge.**

From the foregoing, it seems certain that iridectomy wounds heal and are closed up permanently whenever the iris tissue is not normal. Henderson seems to agree with this statement. When the iris is normal they also heal in the rabbit and have been seen to have healed in the human eye.

If a pathologic condition of the iris tissue is the *sine qua non* for the healing of the iridectomy wound, we certainly should expect that the wound of an iridectomy performed on a glaucomatous eye, except, perhaps, in the very earliest beginning of the disease, is certain to heal, as the iris tissue is never in a normal state. In such early stages the eyes are but seldom operated on. On the other hand, all observers are agreed that the earlier an iridectomy is performed, the better the result. Yet, while this is accepted as a general rule, chronic simple glaucoma is excepted from it, and for this form of glaucoma numerous substitutes for iridectomy have been invented and are being practiced with more or less success.

How an iridectomy succeeds in reducing the abnormal intra-ocular pressure and in curing glaucoma, is a question which undoubtedly has agitated the mind of every thinking ophthalmologist since von Graefe first recommended this operation.

There are so many theories concerning the etiology of glaucoma that it would lead too far to enumerate them in this paper, especially since they are all well known. It seems altogether impossible to reach a definite conclusion. Only one thing seems to become more and more certain—that is, that a pathologic process in the general, perhaps, but surely in the intra-ocular blood-vessels is directly responsible for the glaucomatous state.

In a general way, more fluid must be secreted into the eye-ball than can be carried away, or the nature of the intra-ocular fluids must be so altered that their consistency does not allow them to leave the eye through the preformed channels, even if the latter were normal; or, finally, the means of egress, even with a normal formation of intra-ocular fluids, must become closed by some pathologic process, in order to produce an increase of intra-ocular tension.

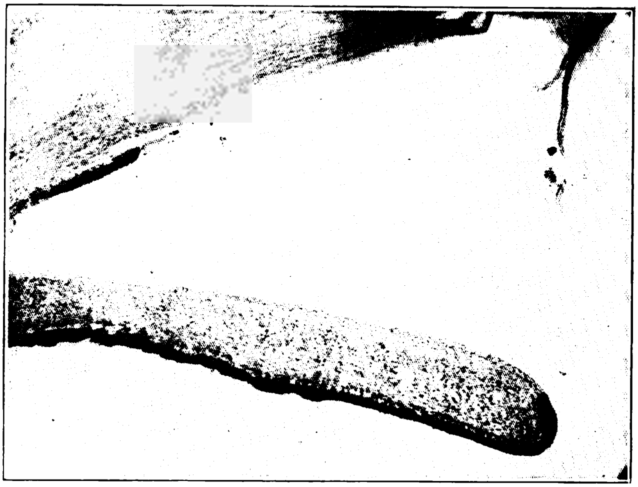

Figure 13

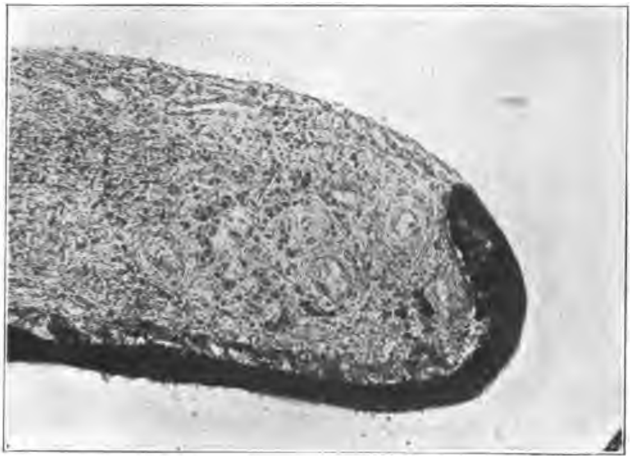

Figure 14

Figs. 13 and 14.—This shows a similar condition to that shown in Figs. 10, 11 and 12. The specimen is from another glaucomatous eye with an iridectomy stump of older date. There is less infiltration in the tissue of this stump than in the foregoing ones. It is almost altogether covered with pigment epithelium. The whole anterior surface of the iris and the stump is covered with an endothelial membrane.

If we still believe that the iris angle is preeminently the place of exit for the fluids from the eye, and I confess to a firm adhesion to this opinion, we must, also, see the main cause of the glaucomatous state in a blocking up of this angle, whether this be due to a mere application of the iris periphery to the ligamentum pectinatum by swelling or pressure from behind, or to a true and firm adhesion between iris and ligamentum pectinatum and later Descemet's membrane, or finally to a plugging of the meshes of the ligamentum pectinatum by cellular or other substances carried there, which on account of their size or on account of their consistency cannot pass out as the normal limpid fluids do.

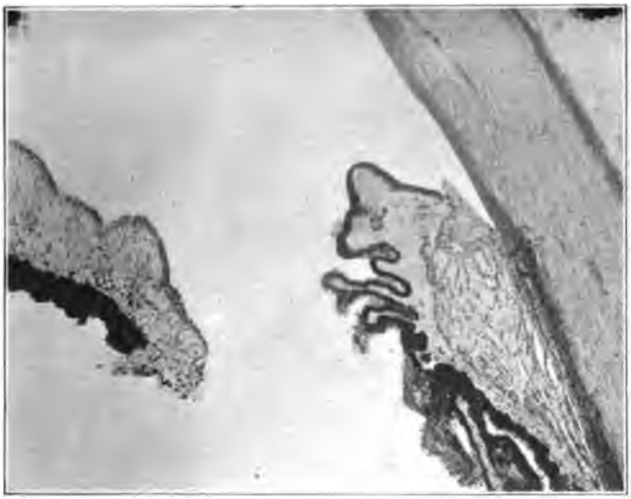

Fig. 15.—This shows the torn edge of the iris which has apparently not healed over. The section is from the center of the iridodialysis where, all blood-vessels being torn, no reaction seems to have followed the injury.

What influence can an iridectomy exert on these conditions? The question is as difficult of solution as the one concerning the etiology of glaucoma.

Henderson accepts[4] the obstruction of the iris angle. Yet, in his explanation of the action of an iridectomy he adduces the unhealed iris wounds of the "normal iris" and says: "Iridectomy effects the formation, so to speak, of a large open crypt presenting three sides of a square, the raw surface of which allows of the free passage of the aqueous to the iris veins. The primary obstruction presented by the sclerosis of the cribriform ligament is thus counterbalanced, etc:" Granted that this is true, what happens when the wound of the iris heals and then instead of a new large open crypt having been formed, the absorbing (?) surface is reduced by the amount of iris tissue removed by the iridectomy?

In another recent work J. Kuschel,[6] who believes that the increased pressure is due to sclerosis of the tissues and to an obstruction to the flow of the fluids from the vitreous body into the anterior cavities of the eye, states that the effect of the iridectomy is due with the greatest probability to a rupture in the zonule of Zinn and the anterior limiting layer of the vitreous body during the operation. This assumed rupture, in his opinion, relieves the tension in the vitreous body and the excision of a piece of iris renders a direct connection between the vitreous and aqueous bodies sufficient to allow the fluids

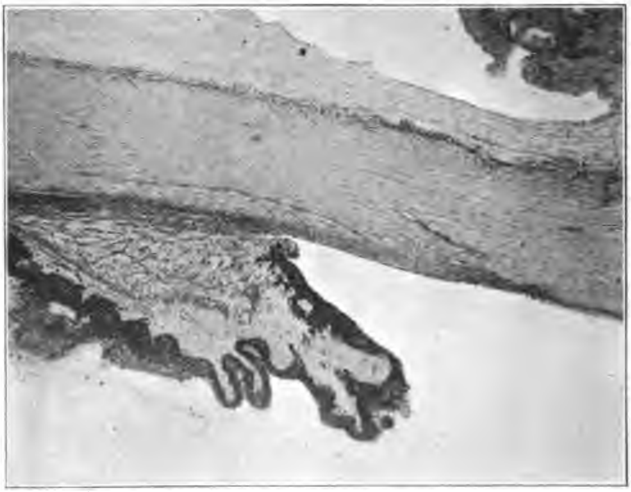

Fig. 16.—This gives a better view of the cyclodialysis associated with the iridodialysis in this eye. The ciliary body is drawn backward from its insertion on the corneoscleral tissue. The little stump made up of the tendon of the ciliary muscle and some iris tissue is well healed over. In this same eye at the time of the injury a further wound was caused in the sphincter edge of the iris, very similar to what an iridectomy might cause at the base of the coloboma. See Figures 17 and 18.

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[6] Der intraoculare Flüssigkeitsstrom in seinen Beziehungen zum biochemischen Aufbau des Auges unter gesunden Verhältnissen, beim Glaucom und bei der Kurzsichtigkeit, p. 123.

to pass out from the vitreous body thereafter. Here, then, the iridectomy appears to be of secondary value only; the rupture of the zonule of Zinn and of the anterior limiting layer of the vitreous body are of prime importance. And yet it seems to be a well-established fact that a rupture of the zonule of Zinn with the consequent subluxation of the lens can give rise to a secondary glaucoma.

Since Lagrange's introduction of his sclerecto-iridectomy, several other methods of making a permanent hole in the sclerotic have been devised, all in the hope of adding to the benefits of an iridectomy that of a filtering scar. But the proof that the resulting scar, if it ever is, will remain a filtering one is wanting, and I am afraid will remain so. That the bulging of the tissue which covers the scleral opening adds still more to the room which the fluids of the eye can occupy is true, but what gives us a right to assume that the conjunctiva covering the hole allows any fluid to filter through? Does a staphyloma of the sclerotic, even if stretched to bursting, let any fluid pass through? I at least have never seen it, although I have examined staphylomata which were so thin that the cicatricial conjunctiva covering the scleral hole cannot be much thinner. And, if the scleral hole reestablishes filtration, why add an iridectomy? The value of the scleral hole, if it remains permanent and covered by a thin tissue layer only, seems to lie in the fact that this thin tissue is more elastic and can yield somewhat to an occasional rise of intra-ocular pressure.

From these and other considerations, and from the fact that iridectomy wounds in glaucomatous eyes do heal, as a rule, the beneficial effect of an iridectomy, it seems, must be due to some thing or things which happen when and while the operation is being performed. We know that in some cases the iridectomy does not reduce the intra-ocular pressure; in such cases this something evidently cannot or does not happen. We know that in some cases even a poorly made iridectomy cures a glaucoma, although the classic broad peripheral iridectomy is looked on as the only correct operation. We have all learned from experience that sclerotomy, much vaunted at one time, is inadequate for the relief of an established glaucoma. The value of the iridectomy with an incision in the corneoscleral margin—if not

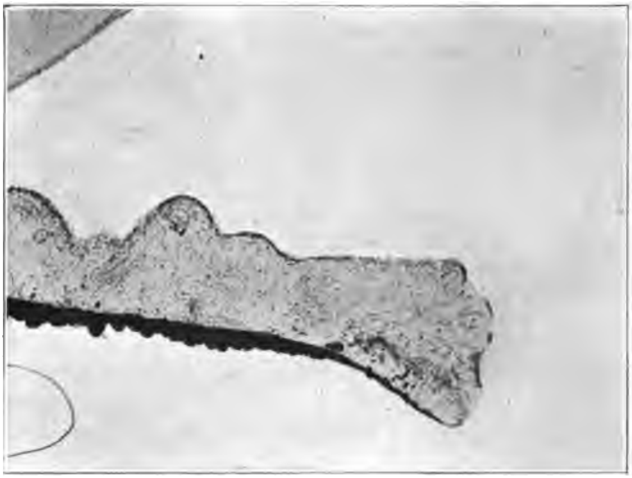

Figure 17

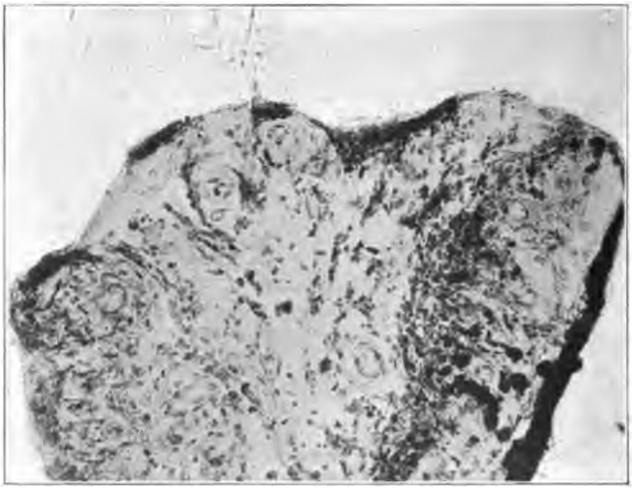

Figure 18

Figs. 17 and 18.—These illustrations show a wedge-shaped gap in the pupillary edge, apparently filled with a similar tissue, but less compact than the old iris tissue, and containing some bloodvessels. It appears, therefore, as if the two flaps formed by this peculiar rent had been united by a wedge of newly formed tissue. The surface of this scar is covered by a layer of anterior endothelium, which varies considerably in thickness.

quite as peripheral, perhaps, as in sclerotomy—cannot, therefore, be due to this incision.

Thus we come back to the probability that, after all, the excision of a piece of iris of sufficient size is the main part of a glaucoma operation. To this may be added a certain amount of reopening of the filtration angle, if only in parts, during the execution of the iridectomy, perhaps, by the direct pull on the iris by which a part of the peripheral iris adhesion may be loosened.

If this is the case, there seems to be only one solution to this question, and this is that the iridectomy, instead of opening up new filtration ways, as Henderson thinks, reduces in reality the secretion of fluids into the eye or alters their composition.

The conclusion thus arrived at through the study of the healing of iridectomy wounds is not only not new, but was the very explanation for the action of iridectomy on glaucoma offered by von Graefe himself. How the excision of a piece of iris, which does not seem to be a secreting membrane, does bring about this result, it is still impossible to say and must be left to future studies.

316 Metropolitan Building.

## MEMORANDA

# THE SIGNIFICANCE OF RETINAL HEMORRHAGE

## BASED ON A STUDY OF THE SUBSEQUENT HISTORY OF ONE HUNDRED AND EIGHTY-SEVEN CASES [*]

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PROVIDENCE, R. I.

The various types of retinitis seen during a practice of more than a score of years have emphasized the fact that the therapeutic measures at our command in this condition are practically valueless. If recovery occurs, it must be ascribed to the recuperative powers of Nature, rather than to any effort of our own. Except in the albuminuric type, the prognostic significance of the disease has not been sufficiently appreciated to warrant the text-books in succinctly stating the fact, or to influence us in caring for the future welfare of the patient rather than devoting our entire energies to the relief of the existing condition.

It was to ascertain whether other than the albuminuric forms of retinitis were as significant of grave termination that I have made a study of 187 cases of retinitis occurring in private practice since 1890. The much larger number seen in my hospital service has been omitted because of my inability to trace the subsequent history of the patients. Specific retinitis has been omitted because of its nature, and because of the fact that it is usually much more amenable to treatment than the non-syphilitic types.

It has been my practice to diagnose my cases under one of six heads:

1. Retinal apoplexy, due to,
    (a) A rupture of the walls of a vessel;
    (b) Diapedesis.
2. Simple retinitis, secondary to retinal apoplexy.

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[*] This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

3. Hemorrhagic retinitis with secondary hemorrhages.
4. Albuminuric retinitis.
5. Diabetic retinitis.
6. Chorioretinitis.

While the classification may be deemed imperfect, it serves to eliminate the forms of retinitis which are not associated with hemorrhages and affords the information desired for the present inquiry.

Text-books on ophthalmology, with few exceptions, pass the subject of prognosis with the statement that "retinal apoplexy and hemorrhagic retinitis are of grave import," "a forerunner of cerebral apoplexy," or "an indication of grave vascular or cardiac lesions," while they are united in the opinion that albuminuric retinitis is fatal within a short time in 70 per cent. of cases. This part of the subject has been ably discussed by various authors, and the statistics of the cases of albuminuric retinitis are offered merely as corroborative evidence of the gravity of the condition, yet the statement of Porter that "all cases of albuminuric retinitis are found in syphilitics," is so abundantly disproved by my own cases that I cannot forbear to call attention to them. The comparatively few diseases which are ascribed as the cause of death in my fatal cases emphasize our present knowledge of the etiology of hemorrhagic retinitis, which may be briefly summed up as follows:

1. Atheroma.
2. Vasculitis and perivasculitis.
3. Increased arterial or venous pressure.
4. Altered condition of the blood found in nephritis, glycosuria, oxaluria or profound toxemia.
5. Traumatism.
6. Reflex disturbances.

All the deaths which have occurred may be ascribed to one of these six causes.

I have records of 240 cases of retinitis, and of these I have been able to trace the subsequent histories of 187.

TABLE 1.—SHOWING OUTCOME IN 187 CASES OF VARIOUS TYPES OF RETINITIS

| Type of Disease | No. of Patients | Living | Dead | Per cent. Dead |
|---|---|---|---|---|
| Retinal apoplexy | 22 | 17 | 5 | 22.7 |
| Hem. retinitis | 47 | 21 | 26 | 55.3 |
| Alb. retinitis | 101 | 26 | 75 | 74.2 |
| Diab. retinitis | 10 | 5 | 5 | 50.0 |
| Chorioretinitis | 7 | 5 | 2 | 28.6 |
| | 187 | 74 | 113 | 60.4 |

TABLE 2.—STATE OF HEALTH OF LIVING PATIENTS

| | Period of Observation | | | | | | Diseases | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | One Year. | Two Years. | Three Years. | Five to Ten Years. | Over Ten Years. | Total. | Usual Health. | Poor Health. | Blind. | Hemiplegia. | Paralysis Agitans. | Epilepsy. | Mental Disease. | Arterio-sclerosis. | Rheumatism. | Total. |
| Ret. apoplexy | 6 | 4 | .. | 1 | 6 | 17 | 10 | .. | 1 | 4 | .. | .. | .. | .. | 2 | .. |
| Hem. retinitis | 3 | 5 | 1 | 12 | .. | 21 | 11 | 1 | 3 | ... | 1 | 2 | 2 | 1 | .. | .. |
| Alb. retinitis | 7 | 9 | 2 | 6 | 2 | 26 | 14 | 9 | 3 | .. | .. | .. | .. | .. | .. | .. |
| Diab. retinitis | 2 | .. | 1 | .. | 2 | 5 | .. | 3 | 1 | 1 | .. | .. | .. | .. | .. | .. |
| Chorioretinitis | . | 2 | .. | 1 | 2 | 5 | 5 | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| | 18 | 20 | 4 | 20 | 12 | 74 | 40 | 13 | 8 | 5 | 1 | 2 | 2 | 1 | 2 | 74 |

I am unable to account for fifty-three. Seventy-four are living and 113 are dead.

So far as may be judged by such a small number of cases, 60 per cent. of all cases of non-syphilitic hemorrhagic retinitis terminate fatally within a few years; while of the hemorrhagic type a much larger number (55.3 per cent.) than is generally believed, are rapidly fatal. Any disease in which more than half of the patients die within a short time is worthy of careful study.

We may gain valuable information by a consideration of the state of health of those now living, and in all cases of which I have not personal knowledge I have gained my information from the family physician.

Of the seventy-four living patients thirty-four, or 46 per cent., are in confessedly poor health, and the similarity between the specific diseases from which they suffer and the causes of death shown in Table 3 warrants the assumption that the mortality there shown will in a short time be increased.

I desire also to call attention to the fact that it is hemorrhagic retinitis which yields the larger percentage of the cases of poor health.

If these statistics are correct (and I have taken great pains in their preparation, in which I have been greatly assisted by my associate, Dr. J. W. Leech), a study of their findings will reveal what I have endeavored to prove, i. e., that aside from the well-known, speedily fatal termination of albuminuric retinitis, not only simple retinal apoplexy, but all the types of retinitis associated with hemorrhages are danger signals, and that our duty lies more in the safeguarding of the patient's future than in the treatment of the present ailment. It offers to life insurance examiners the possibility of forecasting the future of an applicant, and thus determining his value as a risk with more accuracy than by the ordinary methods of examination, and suggests the advisability of insisting on an ophthalmoscopic examination in every case. The comparatively small number of cases, of course, weakens our conclusions, but what is true of the whole is in a measure true of its parts, and further investigations by other observers will, I believe, corroborate these findings. Of the 187 patients 113 (60 per cent.) are dead and thirty-four are in poor health, and only forty (21.4 per cent.) are reported as in their usual health.

TABLE 3.—CAUSES OF DEATH

| | Nephritis. | Cerebral Tumor. | Apoplexy. | Diabetes. | Arterio-sclerosis. | Pulmonary Tuberculosis | Suicide. | General Paresis. | Cardiac Disease. | Rheumatism. | Diphtheria. | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ret. apoplexy.. | .. | .. | 3 | .. | 1 | .. | .. | .. | 1 | .. | .. | 5 |
| Hem. retinitis.. | .. | 5 | 12 | .. | 1 | 2 | 2 | 1 | 2 | .. | 1 | 26 |
| Alb. retinitis.. | 75 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .. | 75 |
| Diab. retinitis.. | .. | .. | .. | 4 | .. | .. | .. | .. | .. | 1 | .. | 5 |
| Chorioretinitis.. | .. | .. | 2 | .. | .. | .. | .. | .. | .. | .. | .. | 2 |
| | 75 | 5 | 17 | 4 | 2 | 2 | 2 | 1 | 3 | 1 | 1 | 113 |

TABLE 4.—DURATION OF LIFE

| Causes of Death | Died within 6 months. | Died 6 mos. to 1 year. | Died 1 to 2 years. | Died 3 to 10 years. | Died over 10 years. | Total. |
|---|---|---|---|---|---|---|
| Nephritis | 38 | 15 | 10 | 10 | 2 | 75 |
| Cerebral tumor | 1 | 2 | 1 | 1 | .. | 5 |
| Apoplexy | 2 | 7 | 1 | 7 | .. | 17 |
| Diabetes | 2 | .. | 2 | .. | .. | 4 |
| Arteriosclerosis | .. | .. | .. | 2 | .. | 2 |
| Pulmonary tuberculosis | 1 | .. | .. | 1 | .. | 2 |
| Suicide | .. | 1 | .. | 1 | .. | 2 |
| General paresis | .. | .. | 1 | .. | .. | 1 |
| Cardiac disease | .. | .. | 1 | 2 | .. | 3 |
| Rheumatism | .. | .. | .. | 1 | .. | 1 |
| Diphtheria | .. | .. | 1 | .. | .. | 1 |
| | 44 | 25 | 17 | 25 | 2 | 113 |

TABLE 5.—AGES OF FATAL CASES OF NEPHRITIS

| | No. of Cases | Average Duration of Life |
|---|---|---|
| Under twenty years | 1 | 2 weeks |
| Twenty to thirty years | 12 | 5 months |
| Thirty to forty years | 11 | 8 months |
| Forty to fifty years | 9 | 30 months |
| Fifty to sixty years | 25 | 18 months |
| Sixty to seventy years | 12 | 20 months |
| Seventy to eighty years | 4 | 27 months |
| Over eighty years | 1 | 2 months |
| | 75 | |

TABLE 6.—DURATION OF LIFE IN FATAL CASES OF NEPHRITIS

One day, 1 case; 2 days, 1 case; 1 week, 1 case; 2 weeks, 3 cases; 1 month, 6 cases; total, 12, or 15 per cent., died within one month.

Two months, 5 cases; 3 months, 10 cases; 4 months, 7 cases; 5 months, 2 cases; 6 months, 3 cases; total, 39, or 52 per cent., died within six months.

Seven months, 1 case; 9 months, 3 cases; 10 months, 1 case; 11 months, 3 cases; 1 year, 7 cases; total, 54, or 72 per cent., died within one year.

Eighteen months, 2 cases; 19 months, 1 case; 20 months, 1 case; 24 months, 6 cases; total, 64, or 85 per cent., died within two years.

Three years, 5 cases; total, 69, or 92 per cent., died within three years.

Five years, 1 case; 6 years, 1 case; 8 years, 2 cases; 10 years, 1 case; 12 years, 1 case; total, 75.

TABLE 7.—DURATION OF LIFE IN ALL CASES

| | Cases | Per Cent. |
|---|---|---|
| Living at end of ten years or more | 74 | 40.9 |
| Died in one year | 70 | 37.4 |
| Died in two years | 17 | 9.0 |
| Died in three years | 4 | 2.1 |
| Died in three to ten years | 19 | 10.1 |
| Died after ten years | 3 | 0.5 |
| | 187 | 100.0 |

Chances of death in 1 year, 37.4 per cent.
Chances of death in 2 years, 46.4 per cent.
Chances of death in 3 years, 48.5 per cent.

Of those who suffered from retinal apoplexy 45 per cent. are in good health, while in the cases of hemorrhagic retinitis but 24 per cent. of the patients are not either dead or afflicted with some grave disorder. Thirteen per cent. only of the patients with albuminuric retinitis are alive and well, and of the diabetic type there is none.

## CONCLUSIONS

1. Three-quarters of all cases of hemorrhagic retinitis either terminate fatally within a few years or the patients suffer marked impairment of health.

One patient died of diphtheria during an epidemic, but was suffering from Bright's disease, and her life would in any event have been of short duration. One died of rheumatism, four from diabetes, and of the two who died of tuberculosis, one certainly did not have that disease when seen, but she died suddenly while returning from Europe and the cause of death was given as tuberculosis.

With these exceptions, all deaths have been due to some disorder of the nervous or circulatory system, and all cases of impaired health are ascribed to the same conditions.

2. The existence of any form of hemorrhagic retinitis is suggestive of present or future disease of either the nervous or circulatory system.

By reference to Table 5, it will be noticed that the average duration of life in albuminuric retinitis increases regularly with the age of the patients. An apparent exception is found between the ages of forty and fifty, but this is due to the fact that the two patients who lived ten and twelve years, respectively, were between these ages. If these are eliminated, the average duration is twelve months. The largest number of cases occur between the ages of forty and fifty.

3. The duration of life in albuminuric retinitis increases with the age of the patient and the younger the patient, the worse the prognosis in point of time.

There have been nine cases of albuminuric retinitis occurring in pregnancy, and of these, two of the patients are dead of acute uremia; one two weeks after parturition at term, the other at four months. Of the seven patients living all are in excellent health; three have passed subsequent pregnancies without recurrence, five have vision of 0.9, or better, and in two there is impaired vision.

When, in the etiology of any disease, reflex disturbance is given as a cause, it is usually synonymous with lack of accurate knowledge. My warrant for ascribing hemorrhagic retinitis to such a cause is found in two cases which I desire to report in detail.

Case 1. E. G., aged 36, a bank clerk, was first seen in 1898 with a myopia of one diopter, which, when corrected, gave him normal vision in each eye. January 28, 1903, he was again seen, and with the correcting glass his vision was 1.0 in each eye. February 2, while at the dentist's a drill perforated the root of a first bicuspid tooth on the right side of his jaw, and breaking, a portion was left in the tooth. Almost immediately there was a blurring of vision in the right eye, and on the following morning when I saw him there was an extensive retinal hemorrhage just above the macula, the vision being reduced to 2/50. The tooth was extracted and the broken tip of the drill was found protruding about 1/8 of an inch. The splash of hemorrhage was soon surrounded by an area of edema, and several spots of exudation appeared in the macular region, which rapidly increased till they presented the appearance of finger-like projections extending into the vitreous, which waved back and forth with the motion of the globe. In June, of that year, this exudate had disappeared, leaving dirty yellow spots, but the vision was not improved. I did not see him again until June, 1906, when to my surprise the vision was again normal. The retina had resumed its normal appearance, save that one of the ascending arteries was obliterated for about two diameters, and a collateral circulation had been established.

This patient was seen in consultation by Dr. Hasket Derby, of Boston, and he expressed his opinion that the retinitis was a reflex disturbance.

A pertinent fact in connection with this case was that an indemnity on an accident insurance policy was denied on the ground that it was not accidental.

Case 2. In 1905, I saw Miss E. J., aged 40, who noticed on the day following a visit to the dentist that the vision of one eye was blurred. She had been having a nerve killed and had suffered a good deal of pain at the time, but did not ascribe her loss of vision to anything connected with her tooth. She presented numerous splashes of hemorrhage in the retina of the right eye, and with the memory of the first case in my mind, I spoke of the possibility of a similar condition. She immediately consulted her dentist, who assured her that there was no possibility of such an accident, but on the third day she insisted on having the tooth extracted, and, with the dentist, visited my office, bringing the offending molar. It showed no evidence of having been injured, but to make assurance more sure, I made a section of the tooth in the axis of the dental canal and found in it a broken piece of steel broach, a quarter of an inch long. The retinitis resulting subsided in about three months, leaving normal vision.

These two cases can hardly be ascribed to a coincidence.

The following case illustrates the point I desire to make.

Case 3. A. B., aged 40, unmarried, was referred to me on account of severe headaches. There was no history of specific infection, the previous condition of health was excellent, and his physician, failing to find an adequate cause for the headaches, as usual ascribed it to the eyes. He had several spots of retinal hemorrhage evidently of recent occurrence, but no impairment of vision. He was referred to his home physician for a urinalysis and I did not again see him for several weeks, when he reappeared with greatly diminished vision and an active neuroretinitis of each eye. He said that no trouble had been found with his urine and that his physician had, in the absence of other symptoms, given him potassium iodid in 5-grain doses, which he had been taking since I first saw him. Evidently cerebral disturbance was present, and a letter was given him to deliver to his physician, stating my opinion that the trouble was due to a cerebral tumor and that the prognosis was bad. He died on the way home while on the train and the letter was taken from his pocket by the medical examiner called to the case.

If there had been an appreciation of the gravity of this case and more attention given to its possibilities, the fatal termination possibly could have been delayed.

I have endeavored to show by these statistics that all types of hemorrhagic retinitis are of serious import; that even retinal apoplexy is significant of impending danger, while simple retinitis with hemorrhage has a mortality largely in excess of that commonly ascribed to it. I have made no study of the pathology, and make no suggestion for treatment, except to urge that attention be given to the future of the patient and not to the present.

117 Broad Street.

# MEMORANDA

# TREATMENT OF PURULENT OPHTHALMIA AND SIMPLE MEANS FOR ITS PREVENTION IN INFANTS*

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AKRON, O.

Any pus producing organism or combination of such organisms may cause purulent ophthalmia, although the gonococcus of Neisser is responsible for a very large percentage, especially of the worst cases. Contrary to generally expressed opinion, I do not believe that the gonococcus or other pathogenic germs, unless extremely virulent, can quickly and successfully attack or destroy normal corneal epithelium. Intact healthy epithelium everywhere in the body, over skin or mucous membrane, presents the most effectual barrier against infection.

A corneal ulcer, the chief danger in these cases, is due either to the extreme virulence of the infection; poor nourishment of the cornea resulting from pressure strangulation, produced by local pressure of the swollen surrounding parts interfering with blood, lymph and nerve function; to direct pressure, a form of traumatism, by the undrained retained and, therefore, more injurious discharges; or to ordinary injuries and abrasions of the corneal epithelium. The two last-mentioned causes, and, to nearly as great a degree, the second, may be forestalled if free drainage is secured without causing any possible injury to the cornea. Therefore, the policy of treatment should be guided by four considerations—the careful, but thorough, use of antiseptics which are of sufficient strength to destroy or inhibit the growth of the infectious bacteria, but which are not too irritating to the local tissues; watchful care over corneal and general nutrition; free drainage; and, especially, avoidance of even the slightest injuries to the delicate corneal epithelium.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

### ANTISEPTIC RECOMMENDED

A bacteriologic examination of the discharge should be made as early as possible, which will be of much service in determining the treatment. In my practice 1 per cent. silver nitrate solution is usually employed, but in virulent gonorrheal inflammations 2 per cent., and, sometimes, depending on the severity, much stronger solutions may be better for a few treatments. In certain mild non-gonorrheal cases 0.5 per cent. is usually efficient. If the Klebs-Löffler bacillus is found, antitoxin should be administered. If the diplobacillus is present, weak solutions of zinc sulphate should be instilled frequently. The organic silver salts have been much extolled, especially protargol and argyrol (Cheney[1]), but the frequent and lavish use of solutions of these drugs, together with systematic cleansing, requiring separation of the lid margins and, therefore, preventing them gumming together, seems to me to explain the results that have been obtained by the employment of these feeble antiseptics (Graefe-Saemisch,[2] Marshall and Neave,[3] Derby[4]). Since the eyelids are so tightly applied to the eyeball the slightly heavier specific gravity of these solutions than of the tears is undoubtedly of slight importance in spreading them. Argyrol solutions do not compare with those of silver nitrate in antiseptic properties and cannot be used clinically with as nice discernment as to strength required. The appearance in all treatments is the same because of the constant dark color of all strengths of argyrol solutions. It is easy to observe the different effects of 0.5, 1 or 2 per cent. silver nitrate solutions, and if employed with caution, and the resulting albuminous precipitate is carefully washed away by a small quantity of any unirritating solution, the antiseptic effect is sufficient without injuring the tissues. Schneider[5] asserts that results do not depend so much on the antiseptic property of the drug used, as on its power to stimulate the production of a bactericidal substance, leukin. My experience with other antiseptics recommended, e. g., mercury, has been too limited to report.

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1. Cheney: Boston Med. and Surg. Jour., clx, No. 9, p. 269.
2. Graefe-Saemisch: Handbood of Ophthalmology, Fas. 8, part 1, p. 261.
3. Marshall and Neave: Brit. Med. Jour., Aug. 18, 1906.
4. Derby: Boston Med. and Surg. Jour., clv, No. 13, p. 342.
5. Schneider: Arch. f. Ophth. (v. Graefe's), lxxiii, part 2.

It is not enough merely to drop the silver solution between the lids, or to apply it to a part of the surfaces of the everted upper lids, which have been the methods ordinarily used, as by these methods the solution does not come in contact with the organisms in the lower and especially in the upper portions of the eyesac. The lids should be separated and elevated away from the eye-ball, so that a lake of silver solution may lie for one-half minute or longer between them, coming in contact with every portion of the conjunctival sac.

### PHYSICIAN'S DAILY TREATMENT

Once each day the physician treats the eyes, holding the child's head between his rubber- or oilcloth-covered knees, the child being wrapped in a sheet fastened by a safety-pin and held by a nurse sitting on a chair opposite. The physician should separate the lids with proper lid retractors, being careful not to touch the cornea. The lids should not only be separated, but they should be pulled away from the eye-ball. Solid lid retractors should not be used, as they are likely to prevent the solution reaching underlying parts of the conjunctiva. The wire lid retractors on the market do not pass far enough under the upper lid. I had longer models made, but they are more difficult to manipulate than tenotomy hooks with smooth rounded bulbar ends. The latter are more readily introduced and more easily moved from side to side. Smooth, blunt-ended probes, properly bent, will serve. The retractor employed for the upper lid should pull it well away from the eye-ball, and be long enough, about one-half inch, to reach to the upper limit of the eyesac. I employ a large tenotomy hook like Green's for the Smith-Indian cataract operation. A smaller one will do for the lower. The hook should form a slightly acute angle with the handle, as otherwise the lid readily slips from its hold. It should be well nickeled and before using should be carefully cleaned with gauze so that no foreign matter will be wiped on the inner surfaces of the lids.

The physician must be careful in using the lid retractors that they do not touch the cornea. The retractor for the upper lid should be used first, placing it under the lid, the margin of which has been pulled slightly away from the eye-ball; the nurse then pulls the lower lid downward with the finger, everting it sufficiently so that the other retractor may be readily introduced under it. Into the sac pulled open by the retractors held by the physician, the nurse, properly instructed, gently pours several drops of the chosen strength of silver nitrate solution, flooding the whole sac, including its upper and lower limits. The retractors should be moved from side to side in the same direction in order that the solution used may get into every portion of the upper and lower parts of the eyesac. After the silver solution has flooded the sac for one-half to one minute, the nurse is directed to wash it away with normal saline, saturated boric acid solution or sterile water. Care should be exercised so that no gritty, undissolved salt may get into the eyesac, as it might injure the corneal epithelium. For the same reason care should be used not to drop solutions on the cornea, especially from a considerable height, but it should be on the adjoining conjunctiva of the lids or eye-ball, preferably on the side toward the nose. The child's face should be tilted toward the side being treated, so that the solution will not run over the nose into the other eyesac. If both eyes are infected the better eye should be treated first.

After the lid retractors are removed good drainage may be secured by preventing gumming of the lid margins, which may be done by dropping on them one or two drops of liquid petrolatum, or by applying sterile petrolatum, or other simple, non-gritty ointment to the margin of one or both lids. When the lids are separated, especially if the infant attempts to squeeze them together as in crying, the margins of the lids are usually everted so that the vaselin may be applied to one or both lid margins without any danger of injuring the eye-ball. It can be applied by means of cotton tightly wrapped on a sterile toothpick.

## CARE BETWEEN THE PHYSICIAN'S TREATMENTS

The care of the eyes between the physician's treatments consists in maintaining free drainage by smearing the margin of either or both everted lids as often as necessary with some simple, non-gritty ointment, e. g., sterile petrolatum, zinc oxid or 2 per cent. calomel ointment. The same result may be more safely secured by dropping on the margins of the lids liquid petrolatum, solutions of argyrol or even olive oil. I much prefer the liquid petrolatum or similar product to olive oil.

This prevents the lid margins gumming together. No accumulation behind the lids should be permitted. They should be gently separated as often as necessary to relieve the discharge. To be certain of good drainage the nurse or attendant should separate the lids every hour or two, making sure that they are not gummed together, and whenever there is a tendency to gumming drop in the oil or argyrol solution or reapply the ointment. If the ointment is chosen, the nurse or attendant should be instructed to use it carefully so as not to touch or rub the cornea, causing a corneal abrasion with probable subsequent ulceration. Pus that has escaped from the eyesac no longer endangers the eye, but may be wiped from the outer surfaces of the lids by moist cotton or gauze; however, no attempt should be made to wipe or wash it from the eyesac. Such unnecessary attempts at drainage are likely, even in skilled hands, to cause abrasions of the cornea, defeating the chief purpose of treatment. I formerly advocated[6] unnecessarily frequent cleansing and irrigations with various non-irritating solutions, exhausting to both nurse and patient, losing sight of the fact that such frequent treatments subject the eyes to great risk of dangerous traumatisms, especially at night, or with poor illumination, or when the attendants are tired or exhausted.

## TREATMENT IN ADULTS

Treatment in adults is similar to that in infants, except that three or four drops of 1 per cent. holocain or 4 per cent. novocain solution are instilled at minute intervals before giving the daily treatment with silver nitrate solution. Cold applications for a short time after the daily use of the silver nitrate solution are soothing and not as dangerous as in infants. If there is much spasm of the lids an antispasmodic, such as morphin, belladonna or bromids, should be administered. Gonococcus vaccine often proves very beneficial in metastatic gonorrheal conjunctivitis due either to endogenous infection or to the presence of toxins, especially where the original infection is of long standing.

In ordinary purulent ophthalmia of adults good results may usually be expected, if once each day every part of the eyesac is flooded with a proper silver nitrate solution, if the lid margins are separated at regular intervals and prevented from gumming together by occasionally dropping on them liquid petrolatum or 25 per cent. argyrol solutions, or by applying petrolatum or other lubricants, and, most important of all, if great care is exercised that nothing be done, such as frequent washing or douching of the eyesac, to injure the delicate epithelium of the cornea, which almost surely will give rise to an ulcer.

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[6] Stevenson: Lancet-Clinic, Nov. 30, 1907.

Other questions that may arise in treating these often difficult cases in both infants and adults, such as the need of canthotomy or scarification and the after-care of these cases by simple astringents, optical iridectomies, etc., have been sufficiently considered by other writers.

Contrary to my former policy of treatment, I now wish to insist that sufficient drainage may be secured by preventing gumming of the lid margins and by their regular separation; further, that frequent irrigations of the eyesac are not only unnecessary, but positively dangerous, since slight corneal traumatisms and, therefore, ulcers are more likely to result. Frequent irrigations, in order to be safe, must be done faithfully and carefully, in a hospital, by trained nurses, conditions not always obtainable under ordinary circumstances for out-patients or when mother and child should not be separated. Although it may be urged that the use of proper hooks, not possessed by all physicians, is dangerous in untrained hands, by the experienced quicker results are more safely secured than by any other method. In untrained hands any method is dangerous, but when hooks are not to be had or their use feared, the daily instillation of the silver solution, the lids being drawn widely apart and moved in such a way that the solution will be distributed as far beneath them as possible, maintenance of free drainage, and avoidance of dangerous washing and irrigating, will nearly always secure good results, although not as promptly as when the hooks are used.

## PROPHYLAXIS IN INFANTS

On the completion of labor the child's eyes should always receive consideration and attention, as a large proportion of cases of ophthalmia neonatorum—Nance[7] claims two-thirds—are not due to the gonococcus, but to other pathogenic organisms likely to be occasionally present in any woman's vaginal discharges. When the mother has leukorrhea, especially gonorrhea, as easily determined by the microscope as a chemical examination of the urine can be made, considering not only the great danger of the child becoming partially or totally blind, but also the various metastatic conditions (McKee,[8] Stieren,[9] Bull[10]) possible and the increased infant mortality, it is certainly a sanitary crime not to use preventive measures. Pregnant women should be instructed to perform daily external cleansing with soap and water and a clean wash cloth, and if irritating or profuse white discharge is present to consult their physicians.

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[7] Nance: Illinois Med. Jour., October, 1908.

*Method.*—After labor is completed and the child removed from the mother, with clean hands and gauze, preferably dry absorbent, the child's face around the eyes and nose should first be wiped clean, wiping in a direction away from the nose. Its eyelids, which should not be opened during this process, should likewise be carefully cleaned. (This does not sterilize the part of the face around the eyes, which is practically impossible, especially with any unirritating safe strength of antiseptic solution.) Next, the lids should be separated and, if there is no pus in the eyesac from an already established inflammation requiring treatment, one or two drops of 1 per cent. silver nitrate solution should be dropped between the outer ends of the lids. This amount and strength of silver nitrate does not require special neutralization so that instillation of salt solutions is not necessary. Washing or cleaning the eyes with any solution, before or after using the silver, is not recommended, as it is likely to injure the epithelium on the cornea and may wash infectious material from the partially cleaned face into the eye or from one eye into the other. The slightest injury to the cornea, easily avoided, may be followed by corneal ulcer and loss of vision or eye-ball. The lips and nose should be wiped free of mucus and the little finger, wrapped with a piece of gauze, should be passed into the child's mouth and any accumulated mucus removed by an outward sweep of the finger. To help prevent subsequent infections from the nose I advise dropping one drop, not more, of 1 per cent. silver nitrate solution into each nostril. The general toilet of the face and body may then receive attention. If there is considerable swelling or reaction after the use of the silver solution, light cloths wrung out of cold water may be applied for fifteen minutes or one-half hour, but are not often necessary. Harmless bleeding occurs rarely and usually is probably due to unnecessary roughness in separating the lids. If there is delay in labor after delivery of the head, some authorities advise immediate attention to the infant's eyes before delivering the body. Very rarely this might be advisable, especially if the eyelids become separated, but usually would not be good practice, as the accoucheur's and nurse's hands are not properly prepared to treat the eyes and might only infect them. The eyes would also be exposed to subsequent infections during labor.

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[8] McKee: Ophthalmology, July, 1909.

[9] Stieren: Ophth. Rec., July, 1909.

[10] Bull. New York Med. Jour., May 15, 1909.

### AFTER-CARE

*After-Care.*—In subsequently cleaning the child's eyelids and around the face and eyes, the nurse or attendant should be instructed to destroy all wipes and not to use any cloths or solutions dirtied with the mother's discharges or that had been used in previously cleaning any part of the child. They should always be instructed to inform the physician—and are compelled to do so under penalty of the law in many states—of any marked or continued discharge from or redness of the infant's eyes, especially if the lids become swollen or gummed together. Although by the use of prophylactic measures the severe and dangerous ophthalmias are almost entirely prevented, it should be understood that, for the first day or two, slight discharge and redness, due to the irritation of the solutions used, are not uncommon, occurring approximately in 10 per cent. of the cases. This silver irritation quickly disappears, requiring only cleanliness and prevention of the lids gumming together. A few cold applications may be helpful. Repeated instillations of silver salts are not only unnecessary, but likely to prove harmful.

The eyelids are nearly always closed during labor, so that on opening the lids whatever germs gain entrance into the eyesac are probably from the eyelashes or on or near the margin of the lids. Therefore, a drop or two of the silver solution placed in the outer portion of the eyesac, and swept across the eye by the closing of the lids, nearly always reaches all the germs present. It is quite different after inflammation is established and the germs have gained entrance, not only into the upper and lower folds of the sac, but into the recesses of the glands.

It is not only necessary that prophylactic measures be used and properly used with attention to detail, but that continual care be exercised that the eyes do not become infected later.

### IMPORTANCE OF OPHTHALMIA NEONATORUM

The number of persons totally blind throughout life from this preventable disease, according to Keller,[11] 25.21 per cent. of those who enter our blind institutions, and fully one-tenth of the blindness in any country, is only a partial index of its evil results. Account must also be taken of the high mortality among these unfortunates, of the disfigured and disabled eyes of a far greater number of others, perhaps totally blind in one eye, scarred on one or both—with defective vision—and, therefore, lessened earning power.

It is estimated that the total annual loss from this disease in the United States is $7,000,000, and that an amount of more than $1,000,000 annually is spent in partially caring for its victims. The community is said to pay an excess of $4,500 during the school life of a blind child. In this and other civilized countries millions are spent yearly to safeguard the public against a few criminals, but even paltry sums are reluctantly spent in preventing diseases and hygienic evils which are responsible for much more misery, sickness and even death, and which impair the usefulness of many helpless citizens. The property rights of an infant or minor are protected by the state. Parents or guardians are not permitted to abuse him, and for the protection and good of the state he is compelled to go to school and is not permitted to do injurious work while of school age. The infant is entitled to protection against preventable blindness and the state ultimately may find it necessary to help stamp out this pestilence, just as it passes other sanitary measures.

In 1906 a committee of the American Medical Association was appointed, with Dr. F. Park Lewis as chairman, to determine ways and means of securing the more general adoption, where necessary, of well known methods of eliminating this disease. In every state the active cooperation of the medical profession, public health authorities and other agencies is being sought. A national movement has been inaugurated to secure the

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[11] Keller: Lancet-Clinic, Feb 27, 1909.

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passage and enforcement of proper laws dealing with these cases in this country, as has been done by most of the European powers; also to bring about a more general, if not universal, adoption of simple prophylactic measures. This has come about through a recognition of the importance of this disease, not only to the individual blinded at birth, before being trained and educated, but also to the state.

### ADVISABILITY OF ROUTINE USE OF PROPHYLACTIC

In this earnest crusade for the more general use of a prophylactic, it is undoubtedly good policy to insist on its routine use in public lying-in wards or hospitals, where these infections are so common, and by midwives who, with their limited knowledge, cannot be expected to form correct judgments. The capable physician in private practice, however, well acquainted with his patient, is likely to insist on his right to determine in each case, a prerogative of his profession, whether the use of a prophylactic is necessary or not. If a vaginal discharge is present, time will often permit a bacteriologic examination to be made which will determine the variety of pathogenic organisms present, any of which may cause ophthalmia neonatorum. The presence of the gonococcus demands prophylactic measures. In cases, however, in which a discharge is present it is easier to employ a prophylactic than to make a bacteriologic examination. If properly and carefully used, it will very rarely cause more than a little temporary redness and discharge, although the advisability of its universal use is still debatable. It is going too far, is unjust and unfair, to say that if infection occurs or even blindness results that it is always due to the neglect and carelessness of physician, midwife or nurse, for the child's eyes may become infected after the proper use of a prophylactic, as from the nose through the nasal duct, from subsequent introduction of infection, or may have been infected sometime before birth, which Stephenson[12] claims is not infrequent (Lewis, Harper and Pease,[13] Nance[7]). However, these professions must bear a large part of the blame for the continued frequency of this nearly always preventable disease.

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[12] Stephenson: The Hospital, March 12, 1910.

[13] Lewis, Harper and Pease: Ophthalmia Neonatorum and Its Prevention, THE JOURNAL A. M. A., March 13, 1909, p. 876.

While the knowledge that a large proportion of blindness from ophthalmia neonatorum is due to gonorrhea in one or both parents, and placing the real responsibility of the child's blindness on them, will be a moral lesson to the public, the fact remains that it is not all due to this cause. The general understanding and belief that infection may occur in any case, gonorrheal as well as non-gonorrheal, will permit of the freer use of a prophylactic without any danger of giving offense by its use. In addition, discussion and dissemination of knowledge concerning ophthalmia neonatorum, over which a mock modesty is otherwise too likely to draw a veil, will thereby be furthered. Morrow says, "Social tradition holds that it is a greater violation of conventional propriety publicly to mention this infection (gonorrhea) than privately to contract it and carry it home to the family." Unfortunately at the present time this is the best reason why, in the movement for the prevention of blindness, too much emphasis should not be placed on its most common cause. A freer discussion of this commonly, but not necessarily, gonorrheal disease will serve as an entering wedge toward a more general discussion and understanding of sex problems.

Physicians will no doubt be surprised to learn that a social service study of 116 cases of ophthalmia neonatorum cared for in the wards of the Massachusetts Eye and Ear Infirmary, October, 1908-1909, showed that only two of the patients had been attended by midwives, and that only temporarily until their physician could be secured, and that physicians had attended all the other cases throughout. As Edgar[14] has said, "The burden of the blame cannot all be placed on the midwife."

At present, however, the necessary midwife. who in poor families serves not only as accoucheur, but often takes the place of nurse and housekeeper, and who attends about one-half the obstetric cases in our large cities, presents a serious problem for solution. Recommendations in regard to her are freely made, requiring better training, prompt notification of births, compulsory use of prophylactic solutions, early reports, on birth certificates, of the use of such solutions, and immediate reports of cases of ophthalmia, with punishment for repeated neglect by fines or withdrawal of license to practice by registration authorities. The medical profession in this day of many false gods must be careful, however, not to encourage unduly too many legislative activities, especially by making them, at least apparently, necessary. Statutes compelling physicians to use certain rule-of-thumb remedies as a routine for certain conditions will never be advisable nor practical. The beginning of such a course by the public, with its attendant penalties on the profession, may be fraught with grave dangers both to the public and to our profession. The proper use of a good prophylactic by a well-trained physician is a safeguard, but, the statute-forced, perfunctory, careless or improper employment of any solution of silver that happens to be convenient, by any one, physician or midwife, who is not clean and properly trained, is mere interference, and is likely to result in more blindness in the aggregate than if not used at all. The campaign against ophthalmia neonatorum will be truly successful in proportion as it remains one of enlightenment and education.

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[14] **Edgar**: New York Med. Jour. May 15, 1909.

Human error is sure to become manifest in the zeal and enthusiasm attending any great or useful reform or propaganda and great care must be exercised that the habitual reformer or yellow journalist does not harm innocent persons or jeopardize this movement by his too frequent indulgence in misrepresentation or exaggeration. Physicians should not permit such mistakes to prejudice them against a good movement, but should do their part properly to further it.

### VALUE OF PROPHYLAXIS

The prevention of blindness from this cause is too important to demand argument, too successful to require debate, too necessary to need exaggeration or overstatement. The value of prophylaxis in these cases, one of the greatest triumphs of modern scientific, preventive medicine, has been so clearly proved by carefully computed statistics of incontestable accuracy, often published, secured by responsible medical authorities in many civilized countries, that members of the profession can ill afford to assume an attitude of indifference or presume to question the usefulness of a prophylactic (Gehrung[15]). The solution is easily employed and can be readily secured, is freely distributed by many state health boards, and conscientious physicians should always have fresh, clear solutions in their obstetric bags. The objections to frequent use of prophylactic measures, slight pain and mild silver conjunctivitis in many children who would not be likely to have ophthalmia neonatorum, are not comparable in importance to the almost never-failing success in preventing this no longer necessarily venereal or shame disease. The great lying-in hospitals in which cases of ophthalmia neonatorum and its results were formerly so conspicuous, very rarely have any such cases since the routine use of some good method of prophylaxis. That some physicians of large experience have not had any cases of severe ophthalmia neonatorum is their good fortune, but only begets a false sense of security for the future. It is not a parallel argument to say that because some people die of appendicitis, therefore, every appendix should be removed. Such a course would be attended with risks out of proportion to the results to be obtained. The prophylactic properly used is practically never dangerous. It is not true that a drop placed in the eye after inflammation has once begun is the same as one used soon after birth. In the latter case, unless inflammation is already established, there are probably only a few germs along the margins of the lids; in the former they are distributed throughout the conjunctival sac in its folds and recesses and will therefore require severe treatment to destroy them. It is difficult to understand the indifference of many physicians to this important subject, about which so much is being written, for many such physicians are men who frankly condemn and recommend prosecution of quacks, Christian Scientists, etc., for their malpractice and vagaries. The time is nearly here when the accoucheur and nurse will be subjected to severe criticism and reproach, whether deserved or not, and considered culpably negligent and censurable in every case in which ophthalmia neonatorum develops, especially if measures for its prevention have been neglected, as nurse and physician are now blamed if proper aseptic precautions for the protection of the mother against infection are not observed.

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[15] Gehrung: New York State Jour. Med., ix, No. 5, p. 205.

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## MEMORANDA

# THE IMMEDIATE REMOVAL OF TRAUMATIC CATARACT *

JOHN A. DONOVAN, M.D.
BUTTE, MONT.

A paper relating to the treatment of eye injuries, to be properly understood, should be confined to specific cases. The impossibility of the universal application of any single line of treatment is well known to all surgeons, but especially so to those most frequently called on to treat eye injuries. In these cases it is often the most experienced who find it hardest to decide on the line of treatment.

Sweet, in his report[1] of 702 cases, says: "Unlike other operative procedures on eyes in which there is uniformity of technic, each foreign body injury must be studied as a separate and distinct condition."

Bowman[2] is quoted as saying: "I have never laid down a method of extraction of cataract universally applicable. I always operated as seemed best at the time for the individual treated."

Another difficulty in obtaining uniform results is the difference not only in classes of people. but classes of injuries. Sweet's report, which probably fairly represents the proportion of cases in the large cities, gives 81 per cent. magnet cases, while my cases are in almost inverse proportion. He gives 5 per cent. lens injuries. Here, among the penetrating wound cases the proportion of lens injuries is several times this percentage.

The accompanying illustrations are stereoscopic photographic reproductions of actual working conditions, which, when viewed through the stereoscope will explain

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

[1] Sweet: Third Series of Cases of Injuries from Foreign Bodies, Tr. Am. Ophth. Soc., 1909.

[2] Wood's Ophthalmic Operations, p. 1155.

the method and kind of injuries likely to occur in mining districts.

When the patient is seen immediately or soon after the injury, after he has recovered from the shock and before the local reaction has made careful work almost impossible, the lens should be extracted. This may be done under a local anesthetic, though a general anesthetic is preferable in many cases. If local reaction is already marked, the ophthalmologist should wait about four to six days when it subsides, then remove the lens before the secondary inflammatory symptoms have become severe. A general anesthetic is necessary at this time. Though the breaking up of the lens and washing it out has been a recognized procedure for some time, a regular cataract extraction, combined or simple, has in many cases been more successful. If there is a large central corneal wound or if the lens is already badly mutilated, cutting it up with a cystotome and washing it out is the operation of choice. Unusual care must be exercised in doing this, as the zonula, if not already ruptured, may very easily become so in these cases, or if the wound extends into the vitreous there is likely to be some mixture of lens and vitreous. To wash this lens matter out, especially when there are fine particles of rock in the anterior chamber, often requires considerable force. Several times I have found it difficult to be certain just what the escaping fluid consisted of.

In children and young people, if there are no positive indications of a foreign body in the lens, a safer and more simple procedure is to extract by suction with a needle. An ordinary hypodermic needle will answer in children. It is introduced well into the lens, care being taken not to pass into the vitreous. If the needle is turned so its opening can be watched during the suction it is under better control.

In older patients or when particles are in the lens, the regular combined or simple extraction is preferable. Though some may, and with apparently good reason, urge against an iridectomy at this time, as a rule I prefer it, especially in severe lacerated cases. As yet I have seen no bad results that could be attributed to this, while several simple extractions would have done better had iridectomy been performed. If there is a foreign body imbedded in the iris, iridectomy becomes necessary. The incision in the cornea should be made as near as

**knife; needle introduced into lens and most of it extracted.**

Stereoscope supplement to presession pamphlet, Section on Ophthalmology, A. M. A., 1911.

Illustrating article by Dr. John A. Donovan, Butte, Mont., on "The Immediate Removal of Traumatic Cataract."

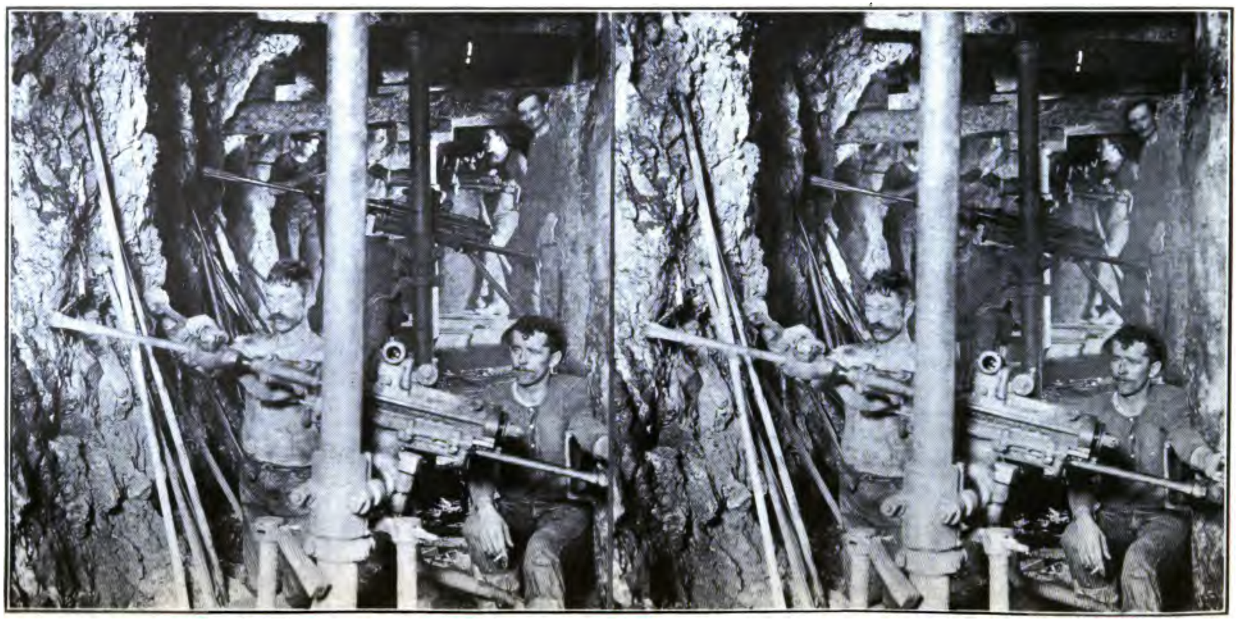

Fig. 1.—Views of miners working on drills, 1,900 feet under the city of Butte, Mont.

.ıe incision in the cornea should be made as near as

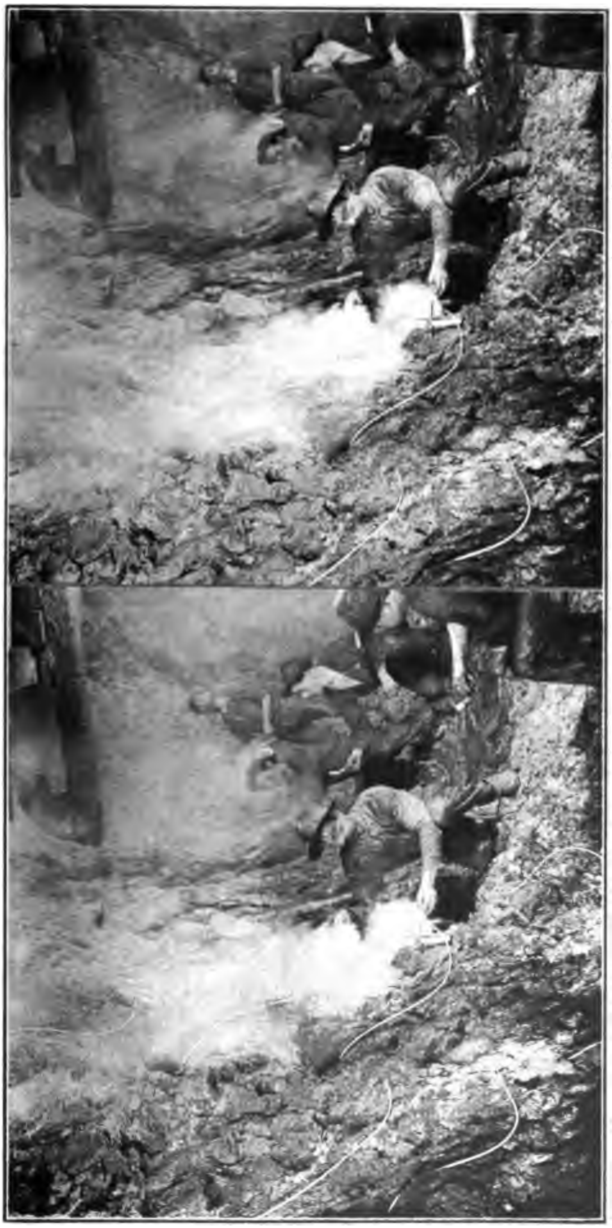

Fig. 2.—Miners ready to light the fuses for blasting in a mine 1,900 feet under the city of Butte, Mont.

knife; needle introduced into lens and most of it extracte incision in the cornea should be made as near as

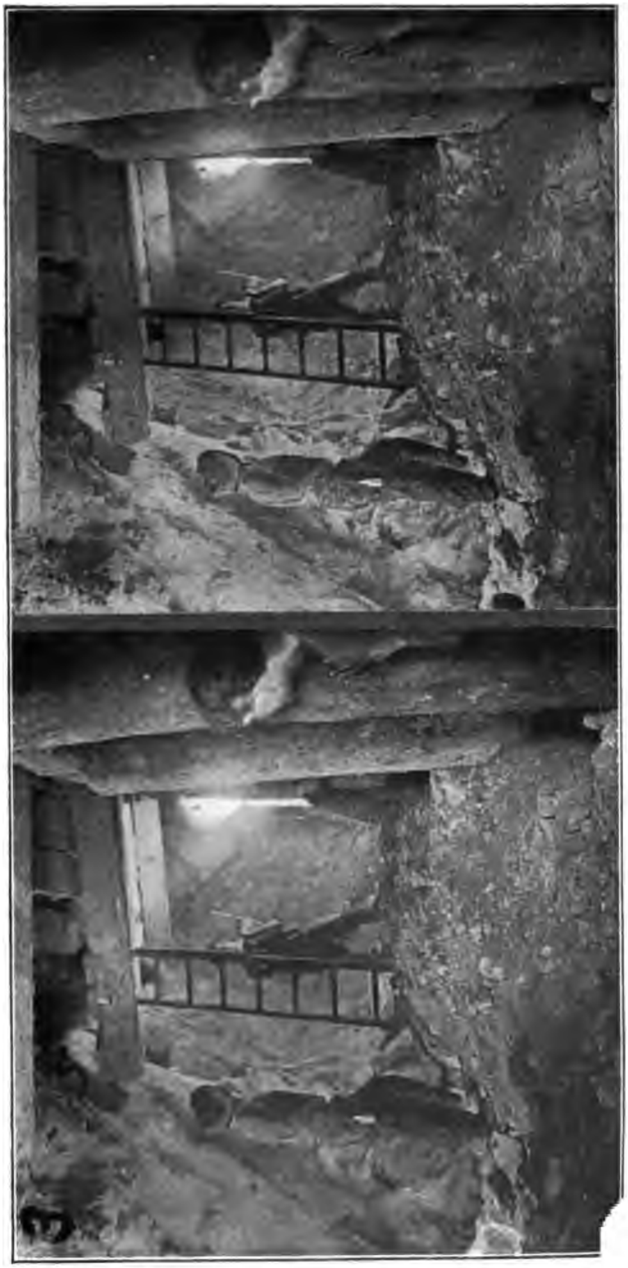

Fig. 3.—After the blast.

knife; needle introduced into lens and most of it extracted.

ie incision in the cornea should be made as near as possible to the imbedded body in the iris. After extraction I fill in between the lids with germicidal ointment (1-5,000 mercuric iodid) using the standard strength if the eye is perfectly clean, or the strong (double this strength), when infection is probable. The formula of this ointment is:

| ℞ | Gm. or c.c. | | | |
|---|---|---|---|---|
| Hydrarg. iod. rub.............. | | 06 | | gr.i |
| Pot. iod. ...................... | | 3 | | gr.v |
| Aquae q. s.................... | | | or | |
| Adeps lanæ .................. | 30 | | | ʒi |
| Petrolati albi............q.s. ad. | 300 | | | ℥x |

It has the advantage of being non-irritating at this time, a powerful germicide not precipitated by alkalies or albumins, thus of decided advantage over corrosive sublimate. Though in a normal eye this ointment may be irritating, in an injured eye, especially during the acute stage it acts otherwise. Besides being a germicide, it prevents adhesions of the lids and dressings and is easily applied. It also prevents that dry sticky sensation. If this does not control infection, 25 per cent. argyrol in many cases does. Of course, one must not rely too much or too long on any particular remedy. The pupil must be kept dilated, the ointment used once, twice or oftener each day and any other treatment as indicated. Salicylates the first three or four days I believe to be of much value.

Case 1.—Man aged 50, blasted December, 1908; each eye contained several pieces of rock in anterior chamber and lens, besides innumerable specks in the cornea. On the fifth day general anesthetic given; the left anterior chamber irrigated to remove loose specks, and then lens cut up with cystotome and irrigated. When about three-quarters washed out, vitreous appeared and operation was stopped. In right eye, all loose foreign bodies were washed out, then iridectomy done and lens extracted by regular combined method. Very little vitreous was lost. Patient left hospital eleven days after operation; was able to distinguish objects in sixteen days. The eye from which lens was fully removed made much more rapid progress and required much less attention. Man is now wearing + 10.00 D. S. lenses and able to make his living working around the mine.

Case 2.—Man aged 25; back fire from rifle caused corneal wound and opague lens. Patient was seen on fifth day; tension high, pain severe, lens much swollen. Twenty-four hours' treatment failed to relieve. Cornea was incised with knife; needle introduced into lens and most of it extracted.

Uninterrupted improvement followed. Vision 20/20 with his glasses.

Case 3.—Man aged 25; steel penetrated lens, entering vitreous; large central ragged corneal wound. The other eye had been enucleated some years before from a similar accident, so it was decided best to take what was supposed no extra risks; magnet extraction, wound cleaned and touched with electro cautery. The swelling of lens with its slow exudation prevented permanent closure of corneal wound for some weeks. There probably was some vitreous exudation as well. It is now two years since injury; eye is shrunken to about nine-tenths normal size; pupil occluded; has light projection. Though an irito-ectomy may restore some vision, this case should be carefully compared with the following one, all conditions being very similar.

Case 4.—Man aged 24, injured same week as above patient, ragged wound entirely across center of cornea. Piece of steel was extracted with magnet from posterior of lens. General anesthetic was given and lens removed by combined extraction operation. Recovery was uninterrupted; patient working in three weeks. Vision after three months with a + 9.00 + 3.00 was 20/30.

Case 5.—Man aged 32; iron in eye-ball, its presence not suspected for eighteen days. Local anesthetic was given, magnet extraction done, and lens cut up and washed out through the same wound in lower part of cornea. In three weeks patient returned home, with eye in nice condition but only light perception and projection as yet. Secondary cataract will be operated on later. As infection was already markedly advanced in this eye, the result was unusually good.

Case 6.—Man aged 53 blasted, seen by me on the fifth day; both eyes full of fine rock. Right eye had rock in lens and cornea. Left eye was still very much swollen, so a thorough examination was impossible. General anesthetic was given and at least five pieces of rock removed from anterior chamber and lens of right eye; lens removed by simple extraction. Patient was taken care of by his physicians, Drs. Brook and Lanstrum in Helena, until able to be moved here three weeks later, when right eye was found in good condition but for secondary cataract and the foreign bodies around the eye-lids. Foreign bodies being now visible in left lens, it was removed by simple extraction under general anesthetic. Recovery slow but satisfactory.

Case 7.—Man, aged 33, came in office while this paper was being completed, injured two hours. Iron 3 by 9 mm. imbedded in posterior of eye-ball was extracted by magnet through lens and original corneal wound. Anterior chamber immediately filled with blood so no attempt was made to remove lens.

The above cases have been selected to illustrate better than can be described the salient points. As a general rule, notwithstanding the extra traumatism, the seemingly increased risk of infection, the removal of an injured lens immediately or a few days after its occurrence is followed by less reaction, less pain, less difficulty in maintaining dilatation of pupil and a very much more rapid recovery than if the case is left to Nature or future operation. In all properly selected cases and especially among the working classes or those unable to lose much time, the immediate removal of the injured lens as above suggested will give the best results.

Those of limited experience in this field, when not certain they are giving their patients the best possible treatment by immediate operation, by all means should not proceed, but treat carefully, study and watch developments as long as they continue favorable.

Uninterrupted improvement followed [illegible] glasses.

Case 3.—Man aged 25; steel penetr[illegible] ous; large central ragged corneal w[illegible] been enucleated some years before [illegible] it was decided best to take what wa[illegible] magnet extraction, wound cleaned [illegible] cautery. The swelling of lens wi[illegible] vented permanent closure of cornea. There probably was some vitreous [illegible] now two years since injury; eye [illegible] tension normal size; pupil [illegible]. Though an iridectomy may [illegible] should be carefully compared with [illegible] [illegible] being very similar.

Case 4.—Man aged 24, injured [illegible] ragged wound entirely across center [illegible] was extracted with magnet from [illegible] anesthetic was given and lens re[illegible] tion operation. Recovery was [illegible] in three weeks. Vision after [illegible] —3 [illegible] was 2[illegible] 30

Case 5.—Man aged 32; iron [illegible] suspected for eighteen days. Mag[illegible] net extraction done, and lens [illegible] the same wound in lower part [illegible] patient returned home, with [illegible] [illegible] will be [illegible] after [illegible] advanced [illegible] this [illegible] the [illegible]

Case 6.—[illegible] aged 3[illegible] [illegible] been [illegible] the [illegible] cornea. [illegible] was [illegible] examination was impossi[illegible] and at least five pieces of [illegible] ber and lens of right [illegible] Patient was [illegible] Landstrom [illegible] later, when [illegible] secondary [illegible] Foreign bo[illegible] by single [illegible] but [illegible]

Case 7. [illegible] being [illegible] ded in [illegible] lens and [illegible] ately [illegible]

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## MEMORANDA

# MEDICAL TREATMENT OF THE PATIENT BEFORE OR AFTER THE CATARACT OPERATION *

D. W. GREENE, M.D.
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It can be said with much truth, that the surgical treatment of the cataract patient has so absorbed the attention of the profession that the medical treatment has been a secondary consideration. Generally, little attention has been given to the patient's physical condition for the most delicate operation of surgery. My purpose is to emphasize the importance of medical treatment for certain types of these patients.

It is a great mistake to underestimate the importance of thorough preparation of the patient for such an operation. It should begin weeks before the operation so that primary complications may be avoided if possible, and that severe intra-ocular inflammations are not likely to follow from what it seems reasonable, for want of a better explanation, to charge to the products of endogenous inflammations, faulty metabolism and auto-intoxication, plus the traumatism of the operation.

A patient with senile cataract is not likely to be in a good state of health, because as a rule, he is advanced in years, leads a sedentary life, and the ravages of disease and the attributes of age have made him a wreck of his former self. Age, *per se,* cannot be regarded as a cause of cataract; but certain nutritive tissue changes—arteriosclerosis and fibrosis—to which we may attribute the oncoming of age, are attributes of this time of life, and these probably predispose to cataract.

The cataract patient should be in a hospital for from twenty-four to forty-eight hours before operation, or long enough to allow the attendant to make an estimate of the physical condition, to unload the bowels thoroughly, to have the urine examined, to note blood-pressure and the condition of the arteries and, if blood-pressure is high, e. g., above 220 mm., to reduce it if possible below 190. The individual should have been on a light diet for several days, and should have had a good night's rest before operation. If nervous, he should have from 30 to 40 grains of sodium bromid, and 10 grains of trional or veronal; or, if his condition demands it, he may be given morphin and hyoscyamin. He should have had as thorough sterilization of the conjunctival sac as is possible.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

For many years, I have used atropin, 4 per cent. solution, in all patients operated on and scopolamin, 1 per cent. solution, if complications arise, or if the former does not break up synechiæ and dilate the pupil, cocain and epinephrin[1] seem to be valuable additions to most mydriatic solutions. In cyclitis, these drugs also do good by putting the muscle at rest. Leeching is of great value in iritis and even bloodletting from the arm may be of use in cases of primary or secondary glaucoma, when the blood-pressure is high and the chamber too shallow to admit of an iridectomy without danger of wounding the iris.

Cataracts, as a rule, come at an age when the vitality of the patient has been reduced and his recuperative powers are far below normal. If arterial changes and fibrosis are the essential causes of aging as is generally believed, the health of an individual so affected will be far below what the operator could wish for, and what he is often powerless to improve by medication.

In my experience, cases of delayed healing of the section are often associated with high blood-pressure. Healing can often be stimulated by the local applications of medicines to the lips of the section, and drugs to reduce high blood-pressure. Sodium nitrate, nitroglycerin and potassium iodid are indicated.

It may be emphasized, that a patient submitting to a cataract operation, should be in the best possible state of health, he should be free from bowel and bladder troubles, cough, dyspnea, dacryocystitis, blepharitis, chalazia, styes, rheumatism, gout, etc. Most of these conditions need only be referred to; their treatment is self-evident. But rheumatism and the gouty diathesis, while of importance before operation, assume their most important rôle as postoperative expressions of the state or condition generally believed to be associated with faulty metabolism and auto-intoxication; to emphasize the importance of these as materies morbi is the burden of the paper.

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[1] For use of this term see THE JOURNAL A. M. A., March 25, 1911, p. 910.

I have had comparatively little experience in irrigating the anterior chamber for removing cortical debris. I believe, however, when much soft lens matter is left to irritate the iris and synechiæ are forming, which mydriatics do not break up, that the section should be opened and the debris delivered by scoop and pressure. I believe it safer to do this even several days after the operation, rather than to treat the iritis and other complications which are likely to follow.

Iritis, cyclitis, and iridocyclo-chorioiditis, if of obscure origin, require more extended notice and careful study as to their cause and best methods of treatment. These will be referred to under rheumatism and the gouty diathesis. Syphilitic, tuberculous or diabetic iritis need not concern us; no one would operate in the presence of such conditions, but any postoperative inflammation which may assume any of the forms referred to, should have careful treatment. In iritis, mydriatics are the sheet-anchor of hope, "the beginning, middle and end of all treatment" (Noyes). Atropin can be used by the nurse, but the physician should always use scopolamin, or duboisin himself, if they are indicated. If the head of the patient is turned to one side so that the drug, when dropped on the cornea, will not come in contact with the puncta, and can be coaxed out at the outer angle with a pledget of cotton, no symptoms of poisoning need result.

I am satisfied, from a considerable experience, that in many operated eyes, which have taken on the conditions of uveal disease, which I am attempting to describe by quotations from authorities and from my own clinical experience, useful vision may be saved by proper medical treatment.[2]

I shall not attempt to discuss at length the chemistry or the perversion of the body secretions or the broad subject of faulty metabolism, diabetes, or the poisonous effect of waste tissue generally as contributory to the failure of some operations. These are largely theoretical problems, of which we know comparatively little; and that little the average operator gains from practical experience rather than from exact knowledge of pathological conditions because, as a rule, he has not had the training in laboratory research to make the necessary investigations. It should be emphasized, however, that the investigation of exogenous and endogenous infections (by those capable of studying them from the biochemical standpoint) is throwing much light on this subject.

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[2] Symposium on Uveitis, THE JOURNAL A. M. A., Sept. 20 and Oct. 4, 1902, pp. 676, 820.

No one can doubt that our most exact and thorough knowledge of faulty metabolism and auto-intoxication and the complications which follow or belong to them must come through laboratory research, yet we are in possession of many empirical facts, whose importance must not be underestimated, which must serve as a working basis of treatment until we have reached a scientific method of treatment.

The medical treatment of these patients offers a field of study which is too often neglected for the greater attraction and opportunity for the display of skill at the operating table. We are prone to forget that all we strive for is not always possible of attainment by the operation alone, but that normal healing is necessary to complete the work of our hands. If the reparative power of Nature fails, the most perfectly performed operation fails.

It is a matter of common observation, that of two cataract patients operated on by the same operator, at the same time, and under conditions as nearly alike as possible, one will do well, while the other may do badly. The surroundings are the same, the instruments are the same, the technic, and all that belongs to the personal equation of the operator is the same, all that can vary is the constitution of the patient, which, as I shall try to show is liable to weakness in digestion. assimilation and excretion. The evils which can result from this triad are believed to be competent to explain the difference seen in the reaction of patients to operations or infections. For example, Axenfeld[3] speaking of metastatic ophthalmia, a somewhat kindred subject, has said:

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[3] Bacteriology of the Eye, page 364.

> Although purulent inflammations only differ from non-purulent in degree, and although we know that the casual agents of the former under special conditions can cause non-purulent, serous, or plastic inflammations; yet the purulent hematogenous inflammations arising in the eye can be put into a special class, for we know with certainty that in every case pyogénic organisms have settled down in the affected part of the eye. We cannot infer in an endogenous purulent inflammation, that the toxins, which were circulating in the blood, have settled down at one spot; the hypothesis that such a microbe-free metastatic ophthalmia exists is quite without proof.

At the Boston meeting of this Section in 1906, de Schweinitz[4] presented a paper entitled "Auto-Intoxication in Relation to the Eye," in which he reviewed in a masterly manner the scanty literature of our limited knowledge of the subject, especially quoting from Albu, who defined auto-intoxication as a poisoning of the organism by the products of its own metabolism. Von Jaksch, Uthoff, Elsching, Weintrand, Alonzo Taylor and others have given similar definitions.

At the Chicago Session in 1908, de Schweinitz and Fife[5] presented another paper, entitled "A Further Contribution to the Possible Relation of Auto-Intoxication to Certain Diseases of the Cornea and Uveal Tracts." These papers, I think, should be regarded as constituting the best exposition of the subject in the English language up to 1908.

In his closing remarks at Boston, de Schweinitz said, quoting from Alonzo Taylor:

> Although we do not know the entity of a single auto-intoxication except the acidosis of diabetic coma, and although we known that no known auto-intoxication is to be attributed to any known end-product of any metabolism, we do know from clinical analysis at least that auto-intoxications exist even if their true nature is as yet a secret. . . . We have reason to believe in the absence of other causes, that under these conditions ocular troubles may also arise largely in the corneoscleral and uveal tracts.

I have copied freely from these papers in order to show how the subject is being developed and the interest manifested in it. It has been shown that lessened alkalinity of the blood from increased formation in the blood or lessened elimination of acids is the essential element in cases of auto-intoxication just referred to.

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[4] The Journal A. M. A., Feb. 9, 1907, p. 502.
[5] The Journal A. M. A., June 20, 1908, p. 2040.

We may ask ourselves, therefore, why the poisons of faulty metabolism may not take other forms and attack other structures of the eye presumably as susceptible to the influence of auto-poisoning as the retina and lens.

With the knowledge we now possess of the auto-intoxicational character of diabetes, diabetic cataract and diabetic retinitis, we may feel reasonably certain that we are standing near the border-line where a number of uveal inflammations, whose cause at best is not well understood, may at any time be shown to be caused by auto-intoxication.

Since these papers were written, much further study has been given the subject, and the little monograph of von Noorden, entitled "Diseases of Metabolism and Nutrition," has been translated into English, and the subject has been brought down to its latest stage of development which is a decided advance, we are told, over what was formerly known. Boardman Reed in the preface to the American edition makes the following statement, which is apropos to our subject:

> Physicians who treat chronic disease successfully must keep a close and intelligent watch on the digestion, excretion and assimilation of their patients. All such will agree with von Noorden: first, that there are numerous forms or manifestations of self-poisoning; second, that the acid forms are among the gravest of them; and third, that those special perversions of metabolism resulting in the excessive production of oxybutyric acid, diacetic acid and acetone which so greatly endanger diabetics and also complicate at times other diseases more or less seriously, are of the utmost practical importance.

Von Noorden himself says:

> Within recent years the idea has become firmly established in the minds of physicians that a variety of morbid phenomena are due to auto-intoxications—are, in other words, attributable to certain poisonous metabolic products. This view, it is true, is not new, for it was familiar to the physicians of past generations and was a part of the teachings of the medical folklore of long ago. It was not, however, until Bouchard and his pupils published their investigations on the subject of auto-intoxications that this theory attained the dignity of a scientific doctrine.
>
> At first we German physicians were by no means inclined to accept the theory of autointoxication that was being so enthusiastically proclaimed. In late years, however, our attitude has become more friendly to the doctrine. This change of front is due to the fact that *a number of toxic products of metabolism have been actually isolated and their mode of*

> ***origin in the organism and their pathologic effect determined to the satisfaction of the former critics of the doctrine.*** **[Italics mine. D. W. G.]**
>
> **We do not, of course, know all we should properly know about the poisonous metabolic products that we incriminate in so many morbid states; but in a large group of important symptom-complexes, we are fortunately in possession of a number of facts that suffice to ground the doctrine of auto-intoxications on a solid chemical basis.**
>
> **The doctrine of acid auto-intoxication in man is founded on the similarity between the most important symptoms of experimental acid intoxications and certain clinical syndromes. We can hardly expect of course to discover the pronounced picture of experimental acid intoxication in each individual case. At the same time, the syndrome of acid intoxication has occasionally been found as a complication of diseases.**

It is difficult to find a definite cause for postoperative non-suppurative inflammation, when the essential elements, which are supposed to enter into the cause, are not well understood themselves. We know from observation that non-suppurative inflammations do occur with and without a wound of the globe, and that more eyes are lost from them, than from suppurative inflammations. This fact alone shows the importance of their study and treatment.

Under some of these adverse conditions we are frequently called on to operate, and the wonder is not that some patients do badly, but that about 90 per cent. of successful operations are possible in the hands of the best operators.

The traumatism of the operation and the open wound are elements which must be considered as predisposing factors. Postoperative diseases, while relatively more frequent, are more complex than those which precede the operation, and like iritis and iridocyclitis, may be extension of the same inflammations still further backward. Plastic inflammations seem to attack the anterior segment of the eye-ball by preference and are recognized as such by the exudates thrown out and by their painful inflammatory character. On the other hand, deep troubles in the postuveal tract seem usually of the serous type, and not so generally painful, probably because of the nature of the inflammation and of the tissues affected. Their importance may therefore be underestimated; but this should not occur with our present knowledge of the seriousness of the condition.

I am convinced, from personal experience, that the comparative infrequence of postoperative complications in Smith's clinic in India is due, first, to the thoroughness of his operation which excludes capsular remains and cortical débris (prolific sources of after-troubles in the regular cataract operations); and second, perhaps of more importance is the diet, age and physical condition of the patients themselves.

The average age of cataract patients in India according to Hirschberg is 42 years, in America the average age is 62½ years, and fat, gouty and rheumatic patients are often encountered in America and are the *bête noire* of ophthalmologists.

Of the agricultural people of the Punjab, who make up about four-fifths of the cataract patients at Smith's clinic, the men are tall, muscular and do not carry any superfluous fat. They live chiefly on a rice-milk diet with a few vegetables and very little meat, and consume very little alcohol. As a rule they work long hours and have insufficient food, and they perspire freely under the rays of the tropical sun. Limited diet and active elimination by the skin are not favorable to the development of faulty metabolism and acid auto-intoxication. Just what influence this may have in preventing postoperative complications we may not know definitely, but the comparative absence of complication following cataract operations among Indians is more than suggestive. On the other hand, among obese and sedentary American patients the conditions which seem favorable for auto-intoxication are more often found, and it is my observation that this latter class furnishes the chief contingent of non-suppurative postoperative troubles.

Why self-poisoning occurs, or in what it consists, or why one person should escape it and another not, under apparently similar conditions we do not know. The fact remains, however, that auto-intoxications exist, and that short, red-faced, thick-necked, heavy-set, fat persons, whose weight is out of proportion to their height, who eat and drink excessively and lead sedentary lives, and who are of the so-called bilious, gouty or rheumatic type are prone to have postoperative reactions and, therefore, are not good subjects for cataract or any other operation. This condition is like that of the alcoholic patient who is likely to do badly when disease or surgical operations tax his lowered vitality. He does not respond readily to treatment, nor does he throw off complications which may arise.

It is not my intention to intimate that I believe that a large or even a moderate percentage of our cataract patients are liable to suffer from uveitis as the results of auto-poisoning.

On the contrary, experience and observation show that the percentage is not large, and that by proper eliminative treatment it can be kept very small. It is to the treatment of this small percentage of cases that I desire more particularly to call attention in closing.

Experience has shown that early, prompt and energetic treatment, long-continued, if necessary, will often save useful vision, which without it, would be lost. There is no room here for therapeutic nihilism. We must have the faith of our fathers in the virtue of drugs and administer them heroically if necessary.

In an operated eye, opened for the first time between the second and fourth days, the lids should be free from redness and swelling, the section should be *firmly closed,* if not healed, there should not be prolapse, incarceration or entanglement of iris, the conjunctival redness and discharge should be slight, the aqueous should not be muddy, the tracery and pattern of the iris should be clear and distinct and the pupillary area unobstructed. Barring later accidents or complications, such an eye will heal kindly with a minimum of reaction.

If, on the contrary, the eye should show any of the following conditions: photophobia, chemosis, excessive redness and discharge, a cloudy cornea, cortical debris, muddy aqueous and discoloration of the iris, with, it may be, a gaping wound or vitreous presenting and iris prolapse and vision equal to hand movements, we know that things are not going well with that eye. In such conditions, we may not be able to recognize any cause for or limitations of these complications, we may not even know whether the infection has come from germs introduced into the eye, *exogenous infection* or whether the condition is an *endogenous inflammation* which by exclusion we can believe is caused by faulty metabolism and auto-intoxication, but the indication to open the flood-gates of the system, the bowels, the kidneys and the skin, and eliminate all the poison possible is imperative.

This is not a large subject, so far as conditions before operations are concerned, because most of these patients have been under the care of the family physician or have consulted the specialist who has already had them prepared for operation. While treatment may be influenced or, in a manner, determined by the condition and health of the patient before operation, the more particular requirements of treatment are for the relief of conditions which are associated with, or follow, the operation in a patient predisposed to inflammatory complications to which attention has been called.

In the absence of any known cause for complication, we may suspect auto-poisoning and give deep injections behind the eye of different forms of mercury, salt solution and pilocarpin sweats or Turkish baths. Urotropin (hexamethylenamin), potassium iodid, colchicum, sodium salicylate, the latter in immense doses, 1 grain for each pound of body weight in twenty-four hours (Gifford).

The use of urotropin (hexamethylenamin) as a preventive of infection while the section remains open, or even after it has closed, has not yet been sufficiently studied. Its utility in these eye conditions is deduced from its known antiseptic action through the urinary tract, and it is also known to be eliminated through the aqueous humor, and is thus thought to prevent or modify a beginning inflammation of the uvea.

I have used it in over 100 cases, although I have not had a purulent infection, I have not reached any definite conclusion regarding its efficacy. Its irritating action on the bladder is an objection to its use, and I have not, therefore, been able to give the large doses, from 30 to 60 grains, recommended by some writers,[6] who insist that these massive doses are essential to success, but have been content with doses of from 7 to 10 grains three times a day.

Hot applications, leeches, atropin, scopolamin, eserin or dionin subconjunctivally, according to the state of the tension and other conditions of the eye-ball, are of great value. The value of mercurial saturation of the patient by the mouth subconjunctivally, hypodermically, intravenously, or by inunction is as well established as any empirical fact we have in medicine, and is the chief reliance of our English confrères in deep seated intraocular inflammations.

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[6] Dinkelspeil: Jour. Ophth. and Oto-Laryng., November, 1910.

As stated, we are in possession of many empirical facts which must serve as a working basis in the treatment of the results of faulty metabolism and auto-intoxication, which must guide us until we have a scientific method of treatment. Postoperative delirium is believed by some to be a result of auto-intoxication. This statement I would not dispute. I think, however, that loss of orientation is more often due to defective cerebral circulation, due to sclerotic arteries and the use of the occlusive bandage, than to any other cause. The relief which usually follows removal of the bandage, is evidence of the correctness of this view.

It may be a question whether the part of the paper which relates to the evil effects of auto-poisoning as it concerns the operated eye, will be regarded as of much importance in itself because of our limited knowledge of the subject. But I am convinced that any one who is sufficiently interested will find that the subject is a broad one with enough known truth pervading it to suggest that further study and research will be rewarded by a broadening and accuracy of our knowledge of the harmful effects of self-poisoning, and the means of preventing this condition, which will reward the investigator and benefit mankind.

19 North Perry Street.

# MEMORANDA

# VACUUM FIXATION OF THE LENS AND FLAP SUTURE IN THE EXTRACTION OF A CATARACT IN ITS CAPSULE *

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No ophthalmic surgeon questions the desirability of extracting a cataract in its unbroken capsule. It is now more the problem of finding an intracapsular method that may be followed by the average operator with equal facility and no more jeopardy to the eye than from the capsulotomy extraction. Skilful operators working under exceptionally favorable circumstances may obtain brilliant results from methods not justifiable for general adoption.

From subsequent experiences with a method of intracapsular extraction which I first described,[1] I believe it includes the necessary ease of performance and the indispensable good results to entitle it to consideration for general use. The steps are as follows:

1. The usual preparation of the patient and eye with the preliminary dilatation of the pupil.
2. Cocain and epinephrin are used for local anesthesia and blanching.
3. The speculum is introduced and section made in the limbus, which should include one-half of its circumference, and raise the conjunctiva above but stop short of severing the flap; a fine silk suture is introduced as shown in Figure 1; then the bridge is cut with one snip of the scissors.
4. Now the iridectomy is performed if a preliminary one has not already been made; a preparatory iridectomy is always desirable.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

[1] Ophth. Rec., December, 1910.

5. The speculum is next removed and if blood in the anterior chamber obscures the pupil it should be washed out with a gentle stream of warm saline solution.

6. The assistant holds the upper lid with a lid hook and controls the lower with the thumb of his other hand. Under no circumstances must he permit pressure on the globe until the eye is finally closed; the patient at the same time should look straight ahead, *never down.*

7. The cup of the vacuum extractor (Fig. 2) is introduced from the side through the section and gently placed on the anterior capsule over the center of the lens. If the pupillary margin is everywhere free the nurse is directed to turn the cock at the gauge. The vacuum cup will then grasp the cataract most rigidly. Now the lens should be slightly lifted and rotated on its anteroposterior axis to sever the suspensary ligament; then with the upper edge slightly advanced the cataract in its capsule is slowly and gently lifted out through the pupil and section.

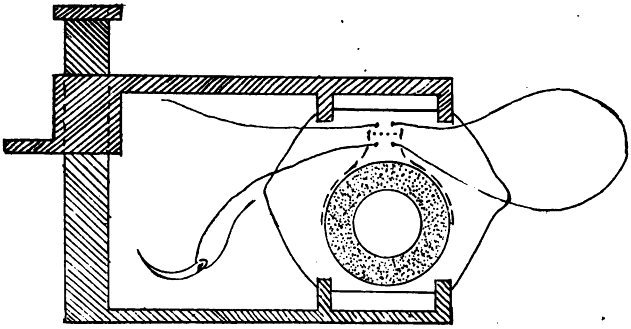

Fig. 1.—Diagram of method of inserting suture in conjunctival flap.

8. The suture is immediately tied. Safe from the loss of vitreous, we may now replace the edges of the coloboma with the iris repositor (introducing it to each side of the knot) and perform the usual toilet of the eye without apprehension.

Particular attention is directed to the advantages of the flap suture tied on delivery of the lens in all cases of extraction in capsule. After suturing the section I was gratified with the ease of iris replacement and safety in all manipulations of the eye. The advantages of this securely closed section obtain throughout the entire healing process. When the cornea is bisected, as is necessary in all forms of intracapsular extractions of cataracts, one must admit the great value of a sutured conjunctival flap from a nutritional standpoint. The presence of this suture well above the cornea seems in no wise objectional.

My first efforts were along the line of making long conjunctival slits by continuing the section upward, but I found it impossible, for lack of room, to deliver the lens thus subconjunctivally with my apparatus. Further experience may perfect the subconjunctival extraction with the vacuum cup, and this perhaps without an iridectomy—an ideal operation indeed.

To remove a cataract in its capsule it has been necessary to either express it by external pressure-manipulation sufficient even in the hands of a careful operator to endanger the vitreous; or to transfix the lens internally with forceps or a sharp instrument, thus necessarily wounding the capsule and thereby possibly freeing the lens and defeating the very purpose of the method; a combination of these two manipulations courts the disadvantages of both methods. Fixing the cataract with the vacuum cup obtains firmer control of the lens than can be done otherwise and without injury to the capsule; it also dispenses with all pressure or manipulation to extract the lens. With vacuum fixation the delivery may be hastened or retarded as desired, and the section may even be "plugged" with the lens should occasion require it.

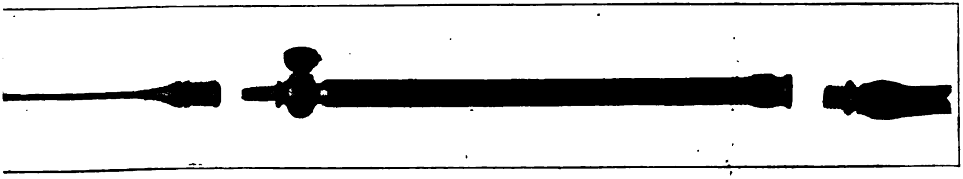

Fig. 2.—The vacuum lens-in-capsule extractor, with handle.

As regards the necessary vacuum apparatus (Fig. 3) but little need be added to the description already published. The cup, if correctly made, is practically no additional tax on the corneal section; the cataract fits into it when the vacuum is connected, so that to make the cup smaller or more shallow would only diminish its "pulling power." The silver shank is malleable but it would be a doubtful improvement to bend it at right angles, etc., as an interested friend has also suggested.

There need be no embarrassment in the introduction of the straight shank extractor and it will not do to endanger the lumen by unnecessary angles. It is important to choose a tube for connecting the extractor to the vacuum which is sufficiently flexible to permit any desired movements in introducing the cup, and yet it must be rigid enough not to collapse from the greatest vacuum pressure required.

It is a problem for the future to determine the precise amount of vacuum for the fixation of cataracts of various consistencies. Too little pressure will allow the lens to slip from the grasp of the cup, and too much may rupture the capsule and disintegrate the soft lens, or even threaten the integrity of the eye. I exhaust the bottle until my gauge registers from 20 to 25. This has been safe and sufficient for the usual senile cataracts.

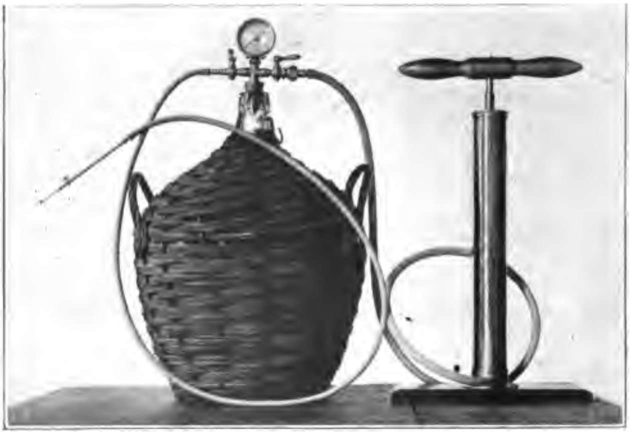

Fig. 3.—The complete vacuum apparatus, with the extractor.

A larger experience and its consideration by various operators should determine the future place of this method in ophthalmic surgery.

# MEMORANDA

## MEMORANDA

# INTRUSION OF THE AMNION AS A POSSIBLE CAUSE OF SOME VARIETIES OF CONGENITAL CATARACT*

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OTTO LANDMAN, M.D.
TOLEDO, O.

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A white disk, called the blastoderm, is the vital part of the egg; after fertilization, it undergoes cleavage and cells form, of which there are at first two layers, namely, ectoderm and entoderm, and then a third layer appears, the mesoderm. The ectoderm forms the skin, central and peripheral nervous system, organs of special sense, and the sphincter and dilator of the pupil.

I shall assume that the blastoderm has been differentiated into these three primary layers, which have spread out and reached some proportions. After a very brief interval the cells of the ectoderm rise up in two parallel ridges, which gradually arch over till their edges touch; then the edges unite and form a long hollow tube, called the neural tube, from which arise the brain and cord, there being at first no distinction between brain and cord. The fore part of the neural tube expands into the embryonic brain, which consists of the fore, mid and hind-brain.

The embryonic eye arises from the fore-brain, as a spherical hollow bud, which gradually swells in its dimensions; this first bud is called the "primary optic vesicle."

A sheet of surface ectoderm lies in front of the primary optic vesicle. The primary optic vesicle, in its growth, soon reaches this sheet of ectoderm, and at the point of contact the ectodermal sheet thickens. This thickened area forms the primordium of the lens. When the optic vesicle touches the sheet of ectoderm its front wall dips in till finally a cup is formed with double walls; an outer, which becomes the pigment epithelial layer; and an inner, which forms the nervous layer of the matured retina. This cup is known as the "optic cup," and has two openings—an external opening, which afterward develops into the pupil, and a slit, which extends from the pupil to the optic stalk, and which is called the "fetal ocular cleft."

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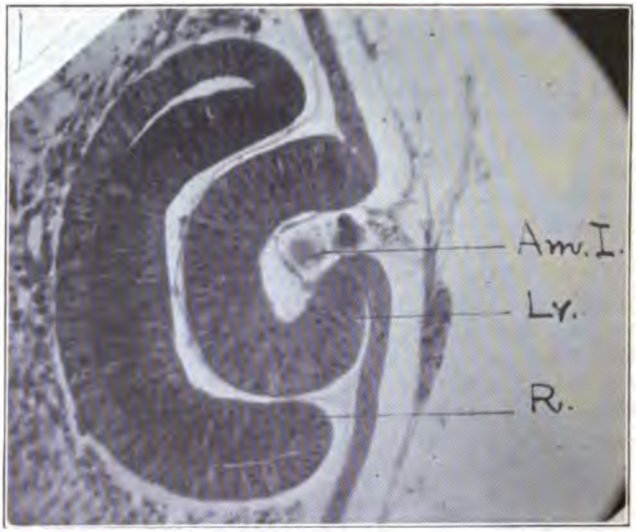
As the outer wall of the optic vesicle begins to dip in, the thickened area of the ectoderm lying opposite the external opening of the optic cup follows it and balloons in toward the sinking outer wall of the cup, and thus a deep pit is formed in this thickened ectodermal area; the pit grows deeper and deeper, till finally its mouth closes and forms a sac, the lens-vesicle, which is still attached to the original ectoderm.



Section of eye of chick embryo. Am. I, amnion intrusion; Lv., lens-vesicle; R, retina.

At length the lens-vesicle becomes detached from the ectoderm and lies in the optic cup. The cells of the posterior wall of the lens-vesicle elongate and eventually the cavity of the lens-vesicle becomes filled and forms the solid lens; finally, the cells of the anterior wall of the lens-vesicle become a single layer—the epithelial layer of the lens capsule.

Now, let us suppose, that before the lips of the lens-vesicle had had time to unite, some form of tissue was thrust in between the lips and cut off from the ectoderm with the lens-vesicle, so that this abnormal tissue lay in the interior of the vesicle, and thus it might interfere with the normal development of the lens and disturb its nutrition.

This possibility is illustrated by the specimen of a chick's embryo, which will be demonstrated, and is described as follows:

> "The specimen is that of a chick's eye. Some tissue can be seen in the concavity of the lens; moreover, the early appearance of this tissue abnormally placed may serve to account for an embryologic explanation of congenital lenticular changes.
>
> "The embryo is that of a chick sixty-nine hours old and hatched at 38 C. It was fixed in Flemming's solution and hardened in the usual way in ascending strengths of alcohol, imbedded in paraffin and cut into series, 7 microns in thickness. The entire eye was cut into sixty-five sections and stained in saffranin.
>
> "Five of the sections show the condition to be described:
>
> "The optic cup has developed normally and the ectoderm has sunken into the cup to form the lens. The lens is in that stage preceding its closure to form a vesicle, and just prior to its separation from the ectoderm. Between its lips dips a part of the amnion with its cells plainly visible and filling incompletely the lens-pit. The ectodermal layer limits the mass and contains two nucleated cells, while a third cell lies on the membrane; within the sac thus formed lie three large spherical yolk masses, deeply stained, surrounded by yolk-detritus. In the region above the lens, what is striking is an excessive and unusual accumulation of yolk-masses, some of which have wandered into the protrusion of the amnion. The ectodermal layer of the amnion can readily be traced outwards, where it is continuous with the amnion proper.
>
> "In Section 26 there is a stage in which the closing lens seems to be in the act of cutting the sac off. This could only be proved by making sections at a right angle to the visual axis of the eye and seeing if the pedicle of the sac were incorporated in the lens tissue. . . .
>
> "The presumptive evidence would justify the conclusion that the entrance into the lens-pit of misplaced amniotic tissue would produce *pathologic changes in the lens* and that in this specimen the protrusion had grown into the lens-vesicle. Furthermore, an amnion protrusion might produce malformations and even *colobomas* of other parts."[1]

An intrusion of the amnion and subsequent pressure of the lens fibers may produce different varieties of congenital cataract, and its presence might cause changes immediately, or so remotely as to induce the juvenile variety.

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[1] Landman, O.: Anat. Anz., 1908, xxxii, 189.

Fusiform, anterior polar and posterior polar, or coralliform cataract may be easily explained by the above-mentioned theory.

Dr. Crampton, on March 17, 1910, described a cataract in a boy 9 years of age, which had a diskiform opacity between nucleus and posterior lens-pole, and in the center a triangle made up of white, dot-like opacities; such a condition might be accounted for by this theory.

Aurel von Szily, Jr.,[2] first reported a specimen of amnion intrusion in the lens-vesicle of birds, but does not state the possibility of such a condition causing congenital cataract.

In the same journal, in 1908, I reported a similar condition and made the following statement:

> The early appearance of this tissue (amnion) abnormally placed may serve to account for an embryologic explanation of congenital lenticular changes.

Over a year after I had published my observation, A. von Szily, Sr.,[3] says that his son, because of the chromatolytic process immediately noticeable in the amnion intrusion, concludes that this heterogeneous portion of tissue is completely absorbed; yet he himself believes that there is a possibility that this little fold of amnion, which after its separation finds itself between the lens epithelia and lens fibers, can directly or indirectly cause an opacity of the lens.

Although I had stated this theory in 1908—over a year before this author—yet he has not given me credit for it.

230 Michigan Street.

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[2] Szily, Jr., A. von: Anat. Anz., Jena, 1906, xxviii, 230.

[3] Szily, Sr., A. von: Klin. Monatsbl. f. Augenh., April, 1909.

## MEMORANDA

## MEMORANDA

# INTRA-OCULAR NEOPLASMS

## WITH A REPORT OF FIVE CASES OF NEOPLASM AND ONE OF SIMULATED NEOPLASM

F. PARK LEWIS, M.D.
BUFFALO, N. Y.

Malignant intra-ocular growths in children are rare, but in the eye-ball as in almost no other part of the body they are encapsulated. If they are discovered early enough, therefore, and enucleation is at once performed, life may thereby be saved. If, on the other hand, their presence is not recognized until the integrity of sclera or cornea has been destroyed and the orbital or extra-orbital tissues has become involved, the invasion of the brain and face with the separation of the cranial bones results in such a horrible method of death that even if the number of cases of retinal glioma is not great, it is worth while to emphasize the characteristic diagnostic features in order that no child may be denied the right of help—while help is yet possible.

The possibilities are that if all cases were reported, their aggregate number might not be so small. Wintersteiner considers that they constitute 0.04 per cent. of all eye diseases. Of 204,612 cases presenting at the New York Eye and Ear Infirmary in ten years, 0.017 were glioma retinæ. It is not at all unlikely that in the country districts or in the small towns remote from large eye clinics, cases which are never reported occur and many more children die than the records would lead one to suppose. The etiology is still uncertain. A number of instances are reported of several cases occurring in a single family, and the assumption has been drawn therefore that heredity exercises a controlling influence. Van Hoffman, Jr.,[1] at the last session of the Heidelberg

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[1] Van Hoffman: Ann. d'ocul., 1910, cxliii, 32.

cells and those of the undifferentiated cells of the three-months fetus bear a close resemblance to each other, and it is more than possible that the developed tumor bears a direct relation to the halted development of the fetal retina, and the glia and ganglion cells may be in Axenfeld's opinion the outgrowth of the undifferentiated cells of the tumor.

While much has been written concerning retinal glioma, no new facts have been deduced from our recent studies which to any degree change existing views as to the nature of these malignant tumors. The purpose, then, of this report is not to add to our pathologic knowledge of these rare growths, although the four described are of sufficient interest to merit special reference; but it is to urge that these rare conditions be brought more frequently before the notice of the general profession, because they occur so infrequently that a physician in general practice may allow the possibility of such a growth to pass wholly out of his mind, and because without distinctive ophthalmologic appliances, the recogni-

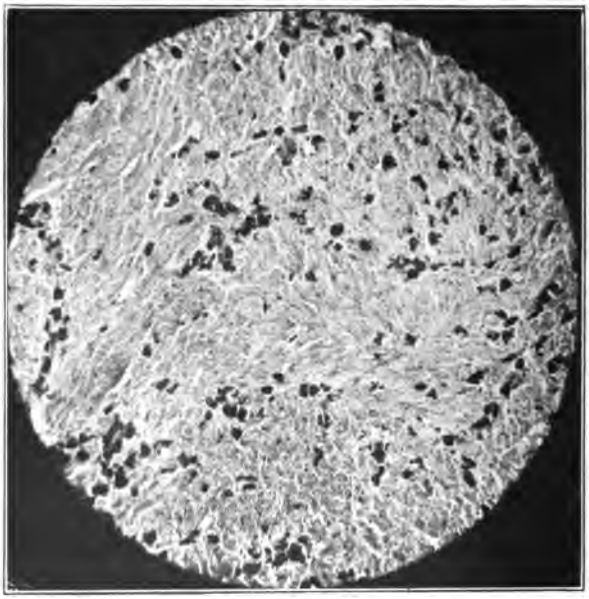

Fig. 3.—Case 2; glioma retinæ. Closely massed round cells, large nuclei; no rosettes found.

tion is so easy on the part of any intelligent observer, and because by its early recognition life may be saved in a large proportion of cases.

Every case should, therefore, be reported and if possible a section of the tumor made and the character of the neoplasm determined. We shall in this way not only get fuller statistical records as to the actual number of cases which occur, but we may, by calling attention to its manifest characteristics, render it possible to make a diagnosis before the growth has passed outside of the eye-ball and while enucleation may yet save the life of the child.

The yellowish reflex coming from the depths of the vitreous lead to the common name given to it by the earlier writers of "amaurotic cat's eye"; and if a light is allowed to shine obliquely into the large pupil, the whitish reflex coming from behind the lens together with the age of the patient is almost absolutely characteristic. When such cases present themselves, skilled advice, if obtainable, should be immediately sought. It is quite different in appearance from the whitish color of a cataract or the surface opacity of a leukoma. It may, indeed, be confounded with plastic deposits in the vitreous from the uveal tract, but these are rare in young children; and when they occur, the usefulness of the eye has probably already been lost.

The first three cases reported below are interesting as being typical of this form of neoplasm. The fourth, that having its origin in the optic nerve, is exceedingly rare. All of these cases occurred, as glioma almost invariably does, in children under 4 years of age. The fifth, that of melanosarcoma, was found in a boy of 13. At present there is no evidence of a recurrence. The sixth is of interest as being the result of an uveitis, possibly prenatal, developing a glaucomatous condition of the eye and simulating in appearance an intra-ocular tumor. An exact diagnosis was impossible until the eye had been removed and sectioned. The illustrations give more clearly than any verbal description the exact conditions which were found in the first five cases.

## REPORTS OF CASES

**Case 1.**—Glioma retinæ; enucleation; no recurrence after nine years.

*Patient.*—A. F., of Pembrook, Genesee County, N. Y., the daughter of simple, honest, hard-working parents, was brought to my clinic on Nov. 26, 1910. She had nearly reached four years, and although there had been some pecularity noticed in the right eye since she was 1½ years old, it was only during the past four weeks that a marked difference was observed by the parents.

*Examination.*—The child manifested considerable uneasiness, pulling her hair on that side and occasionally manifesting evidences of discomfort in the eye. The pupil was dilated and maximum and immovable. The cornea and lens were perfectly clear, while a lobulated whitish yellow mass could be seen apparently filling the posterior chamber. It was traversed by fine vessels, giving in places a light reddish reflection. Tension was apparently slightly increased. There was no response to light, and the eye was evidently perfectly blind.

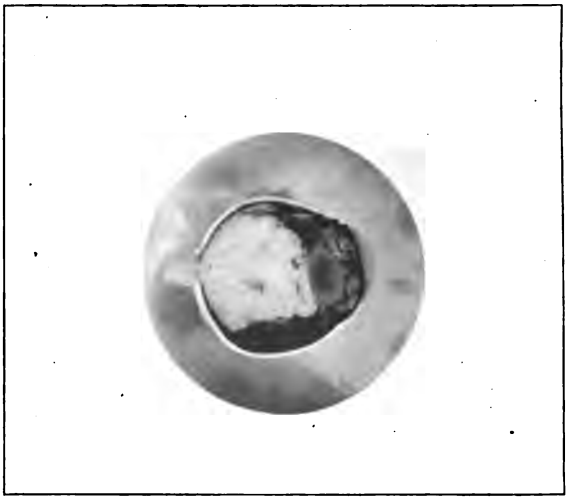

Fig. 4.—Case 3; glioma originating at margin of the optic disk. Beginning staphyloma. Enucleation. No recurrence after eleven years.

The child was apparently in robust health and seemed to be unusually well developed.

*Course of Disease and Operation.*—The diagnosis of glioma was made and an enucleation advised. This was refused for the time, however, by the parents, and the child did not appear at the clinic until April 3, 1902. The tumor had then markedly increased, the eye-ball was flushed, injected and painful, tension plus 2, the lens pressed forward toward the cornea but still clear and in the upper portion of the mass, which was discernible when seen by reflected light, evidence of hemorrhage which had partially absorbed. It was no longer safe to postpone operative measures, so the child was taken into the hospital and an enucleation immediately performed. The eye was hardened and on section of it the diagnosis of glioma was confirmed. Figure 1 shows the extent to which the growth had filled the eye-ball. On April 15, 1911, the child was again brought to my clinic, when the orbit was found to be in perfectly normal condition and there was no evidence of any return of the growth, so that it may be considered to have been permanently cured by the excision.

Case 2.—Glioma retinæ; enucleation; no recurrence.

*Patient.*—C. S., the little daughter of well-to-do parents from another town in Genesee County, was brought to my office by her grandfather, a well-known physician of that place, on Feb. 15, 1908. The little girl was 3 years old. The previous summer she had fallen and struck her nose and from that time a glassy look appeared in the left eye, which seemed to diverge slightly. The day before bringing the child to me, the parents had covered the right eye and had discovered to their alarm that the child was unable to see from the left eye. The little girl had gray eyes, both pupils being normal and both reacting to light readily. There was no history of cancer and the family are quite sure that the child had been able to see out of the left eye for some time after she was born. Lately she had been running against objects and seems unable to guide herself. For the past week she had been unusually irritable.

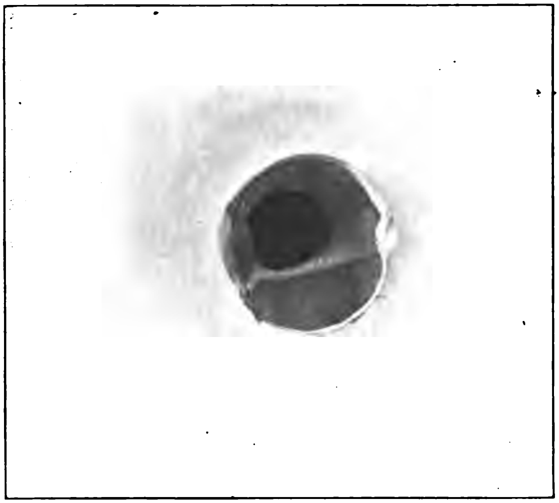

Fig. 5.—Case 4; melanosarcoma; enucleation; no recurrence after a year.

*Examination.*—The eye-ball showed no apparent redness externally; the cornea and lens were both clear, but in the vitreous, flocculent masses that seemed of a creamy whiteness could easily be seen by oblique light without the aid of the ophthalmoscope.

*Course of Disease and Operation.*—The diagnosis of glioma was made and assurance given that within a short time the eye must be removed. It was, however, two months before final permission was obtained. Then tension had rapidly become increased, the eye being injected. The child was immediately taken to the hospital and the eye-ball removed, the optic nerve being divided as far back as possible. A section of the eye was made after it was suitably hardened, and Fig. 2 shows that the vitreous had become completely invaded by the gliomatous mass. Had enucleation been much longer delayed, the growth would have passed through the ocular structures, involving the orbit, when an exenteration of the orbit would have been required, and even that might not have saved the child's life.

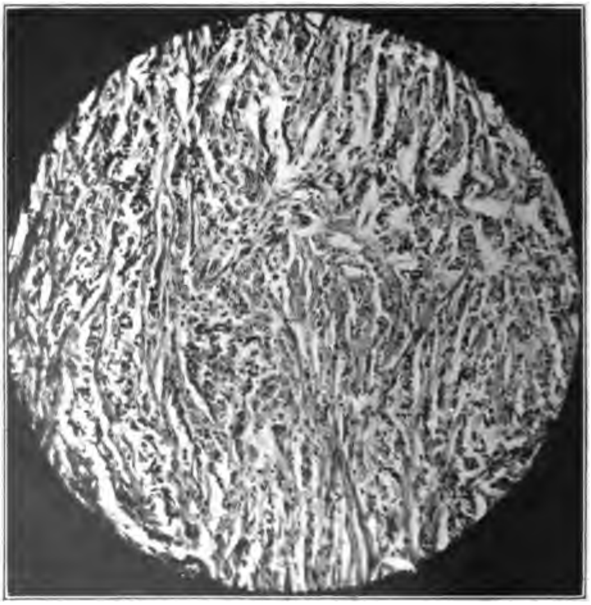

Fig. 6.—Case 4; melanosarcoma. Coarse masses of pigment interspersed with spindle-shaped cells, originating in the choroid.

Case 3.—Glioma retinæ; enucleation of eye-ball; no recurrence after eleven years.

*Patient.*—F. S., of Buffalo, was brought to my clinic at the Charity Eye and Ear Hospital on Oct. 16, 1900, aged 2½, one of eight children. The family was wretchedly poor, often lacking the necessities of life. The youngest of the family, a baby of 6 months, had had spasms from birth. F. had twice had measles; the left eye during the first attack had become swollen with much mucopurulent secretion. He had had a second attack last winter, at which time the eye had become inflamed but apparently had recovered again. The eye began to have a "hollow look," as described by the mother. Three weeks before I saw him, the child had fallen down-stairs, after which the eye-ball began to swell; and during the last three weeks, much of the time the child had held his hand to his head and given evidence of suffering.

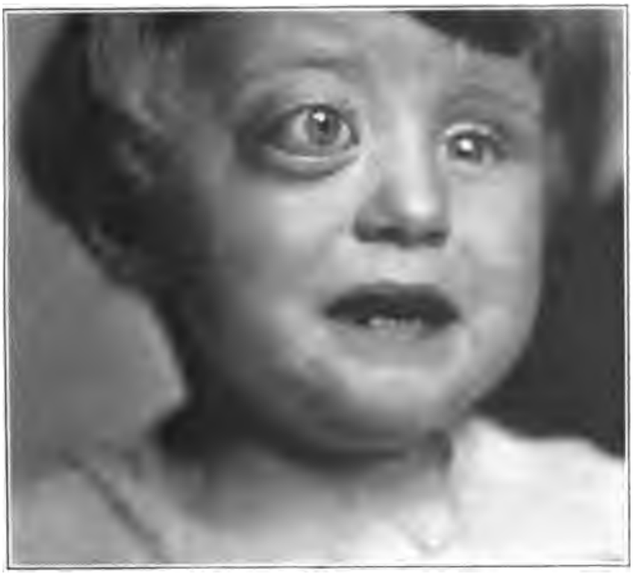

Fig. 7.—Case 5; extradural glioma of optic nerve; enucleation of orbit. No recurrence afterward, although probable later.

*Examination.*—The cornea and lens were still clear, but a yellowish mass tinged with pink filled the vitreous. The red fundus reflex could still be seen around the margin of the enlarged pupil. The pupil did not react to light and tension was increased.

*Operation.*—An immediate enucleation was advised and made. The diagnosis of glioma on section was confirmed and the mass was found already occupying the vitreous space.

Case 4.—Melanosarcoma in boy of 13 years; enucleation; no recurrence. As differentiated from these cases, all of which seem to have originated in the optic nerve, and in none of which was the patient older than 4 years, this case is of interest and value.

*Patient.*—C. B., of Falconer, Chautauqua County, New York, a schoolboy of 13 years, was brought to my office by his parents, who gave a record of good heredity. The patient had, however, not been seeing from the right eye for some time.

*Examination.*—In this eye (the right) he was found to have light perception, recognizing only moving shadows, with entire less of the temporal field, while the light could be perceived on the nasal side. In the left eye, with a plus .50 D. Sph. plus .50 D. Cyl. axis 180, vision equaled 20/20; external chorioidial degeneration with scattered pigment masses was found to be present. A grayish elevated mass was discovered on examination with the ophthalmoscope, occupying nearly a third of the nasal side and leaving a wide margin which was undisturbed between the growth and the optic disk.

The diagnosis of malignant neoplasm was made and the parents assured that enucleation must necessarily be made. The eye, however, was kept under observation for nearly a year before it was removed, when the growth which is shown in Figures 5 and 6 was shown to be a melanosarcoma.

Case 5.—Intradural glioma of the optic nerve; exenteration of the orbit. Glioma of the optic nerve is exceedingly rare. The report of the following case will be of value.

*Patient.*—Baby S. was brought to my office by Dr. William M. Mehl, of Buffalo, for consultation because of a marked exophthalmos. She is the little daughter of parents in moderate circumstances living in Allegheny County. One year previously, the mother noticed that the child frequently rubbed the right eye and shortly afterward it was seen to protrude. This protrusion had continued, slowly increasing to the time of examination.

*Examination.*—There was no evidence of pain and the child was well nourished. All the movements of the eye were still retained and notwithstanding the excessive proptosis, the child was still able to close the lids completely over the ball. It would seem that the lids had grown with the ball so as to accommodate themselves to the excessive protrusion. The eye could not be returned to the orbit with pressure, thereby excluding the possibility of an orbital vascular growth as the cause of the protrusion. There was no pulsation of the eye, nor was it apparently irritable or reddened. The pupil responded to light slightly. The vessels in the fundus were dark and tortuous, and a view of the disk, as far as it could be obtained in a nervous, unsteady child, showed a woolly and uncertain outline. The possibility of a gliomatous involvement was not considered because of the great rarity of tumors of this kind and it was thought that possibly a postauricular abscess or cyst might be causing the protrusion. The child, therefore, was placed under chloroform and an opening was made by the side of the eye-ball and the finger inserted, when it was found that a large roundish growth, apparently coming from the optic nerve, occupied the larger portion of the orbit. Incision was made into this growth, when it was found filled with a translucent honey-like yellowish mass, the tumor being separated by membranous septa. It was evident, therefore, that the growth was gliomatous.

*Operation and Course of Disease.*—The entire mass was removed on Aug. 13, 1910, by Dr. Mehl in my presence. Figure 8 indicates the character of the substance which was found to constitute the tumor. So extensive was the involvement of the optic nerve that the entire exenteration of all of the tissue in the orbit was made. It seemed inevitable that the growth must recur and an unfavorable prognosis was made. In March, 1911, however, the child was again seen and the tissues of the orbit had filled in apparently normally. The lid was not greatly sunken, and there was no evidence whatever of malignancy in the new tissue. It is quite possible, however, that ultimately the growth may recur.

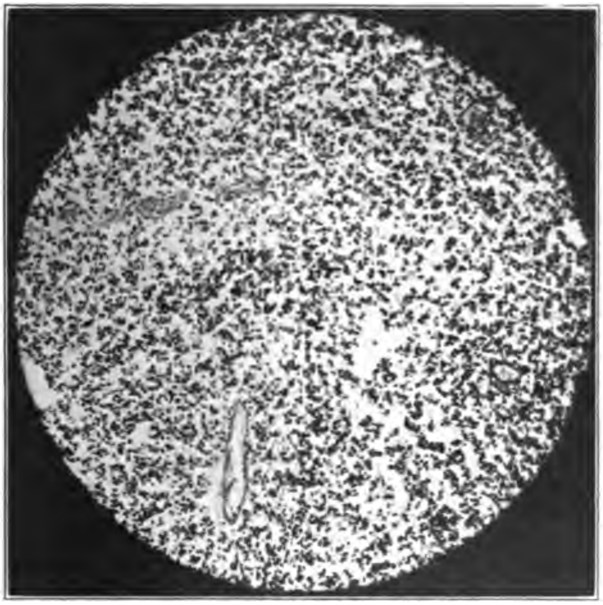

Fig. 8.—Case 5; extradural glioma of optic nerve.

CASE 6.—Iridochorioiditis with occlusion of the pupil simulating an intra-ocular neoplasm.

*Patient.*—E. H., of Fillmore, New York, was referred for consultation by Dr. S. Mitchell of Hornell, in January, 1910. From the October previous the child had been suffering with the right eye. One night a sharp pain developed in the eye and the next morning the eye-ball was very red and swollen. The patient was not seen by Dr. Mitchell for some months; then the eye was found deeply injected, the pupil contracted and filled with a plastic gray mass, and lens and iris pushed forward until it was in contact with the cornea. The eye-ball was intensely hard and evidently exceedingly painful. Dr. Mitchell had advised enucleation. The eye was evidently entirely blind.

*Examination.*—The conditions present when the boy was brought to my office were quite the same as those which Dr. Mitchell had found with the exception that they were intensified. The grayish mass in the pupil had recently become more marked. Glaucoma following a general uveitis occurring in a child three years old and involving only one eye, all led to the suspicion that a glioma was present and was rapidly developing.

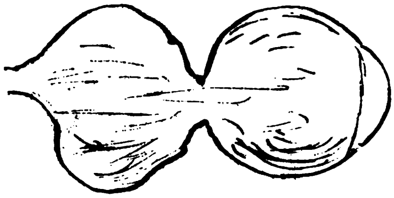

Fig. 9.—Case 6; spider cells from glia.

*Operation.*—The eye-ball was removed by Dr. Mitchell and a section made, when it was found that there was no tumor in the eye whatever. The glaucoma was the result of the obstruction of the filtration angle and of the intense iridocyclitis, which in all probability had existed for a long time before Dr. Mitchell was consulted.

The case is important as one simulating an intraocular tumor and showing the great difficulty in diagnosis prior to enucleation. Happily in either instance, the eye had to be removed and diagnosis was one of secondary, although scientific, interest.

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# MEMORANDA

# BRAIN TUMOR *

## REPORT OF NINE CASES

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SHERMAN VOORHEES, M.D.
ELMIRA, N. Y.

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The following cases of brain tumor seem to me to present variety enough in their symptom-complex to warrant reporting, and to serve also as a basis to direct attention to some of the clinical manifestations present. It has been my experience that some cases of brain tumor are overlooked, especially when no fundus changes have taken place, and that we should not depend too much on choked disk in order to say whether or not we have to deal with a cerebral neoplasm. Especially I think that this is so with many of us who are located in the smaller cities, where we have no expert neurologist to work out with us these central problems. Those of us so situated must depend largely on our own findings for our diagnosis. These cases are much more difficult when they present but few, if any, localizing symptoms outside of the eye changes, and also when the nerve change is in its first or second stage; or perhaps when it never passes on to swelling enough so that it would be classified as choked disk. I am fully convinced that some cases of brain tumor are missed in diagnosis because of the non-recognition of this eye change in its early stages; and sometimes because coincident with the beginning choked disk there appears a macular change; and perhaps some albumin in the urine, with or without the presence of casts, or other evidences of nephritis, and the conclusion is rapidly reached that the case is one of albuminuric retinitis. While the macular figure in brain tumor is not like that of typical albuminuric retinitis, still it is sometimes confused with it; but as albuminuric retinitis

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is usually a late symptom in nephritis, and the macular change in brain tumor comes usually with the height of the swelling, there should be no difficulty in differentiation.

Both nephritis and brain tumor, however, occasionally occur simultaneously, and it is well to bear this in mind; but when there are other symptoms of tumor together with the eye changes it simplifies the diagnosis.

With Dr. de Schweinitz and others, I think that the symptom of temporary amaurosis is of great value, and occurs in the majority of cases. I wish also to emphasize Dr. de Schweinitz' advice to deduct the amount of static refraction present before giving the amount of swelling of the disk.

I believe also that in a majority of the cases the greatest swelling occurs on the side of the lesion, but the exception to this occurs so frequently that as a sign it cannot be depended on in localization. Taken in conjunction with other symptoms, however, it is of value. The same, I think, may be said of nystagmus. While this may be exaggerated when the eyes are turned to the side of the lesion, still it so frequently happens that the contrary is true that it is not of such value as we formerly thought; and, further, nystagmus is far from a constant symptom in brain tumor, and hence loses much of its importance as a diagnostic symptom.

A symptom which I believe is of great value is deafness in one ear, either partial or complete; in the majority of cases of tumor of the cerebellum and angle this appears at an early date. In some of my cases it was among the earliest of symptoms, and is an important factor in localizing the side of the lesion. In the cases here reported four of the patients had deafness on the side of the lesion, as confirmed by operation or autopsy.

The character of the vomiting is usually expulsive; it comes on without any previous nausea, and often is nocturnal. I believe that ophthalmologists should be as familiar with the symptomatology of brain tumor as the internist or surgeon; as ordinarily no one symptom can be relied on in making a diagnosis, and not infrequently the patient consults the ophthalmologist first for temporary amaurosis, or the failure of vision resulting from optic nerve changes. It is interesting to note what a loss of vision occurs in cases which are not yet in the choked disk stage, and in which only a papillitis exists;

and to note also the amount of swelling which may exist in a nerve-head with good central vision.

I believe, with others who have written on this subject, that even if we find a case late in the disease, in which atrophy has already begun, we should not hesitate to recommend operation by decompression and removal of the mass if possible. In this way we are likely to save what vision remains, even if that be but light perception. Another point in this connection is that while saving the remaining vision, operation also serves to lessen the amount of staggering and vertigo, and makes it possible for the patient to get about readily, as Case 3, here reported, shows. In many cases also it serves to relieve nausea, loss of taste and smell, and other symptoms. So sure am I that such advice is for the best interest of the patient that in such cases I always recommend consulting a cranial surgeon. I have seen patients who were blind, deaf in one ear, unable to walk or stand, or to talk so that they could be understood, or to swallow without serious choking, and who suffered severe pain and a constant nausea and inability to retain food, with other of the terminal symptoms that tend to make a patient so absolutely helpless and wretched that nothing could be worse. To the laity, however, a cranial operation is a terrifying thing, and several of my patients refused operation and underwent all these things because they or their families were not educated up to the point where they could conceive of a surgeon doing an operation on the brain and the patient surviving.

I have no doubt that the reversal and interlacing of the color lines as described by Drs. Cushing and Bordley as an early diagnostic sign is of value. I have two cases at present in which these phenomenon occur. If it is found to be constant prior to the visible nerve changes it will be of great service in diagnosis.

In my cases I have not noticed the distention and change in color, and uneven tortuosity of the retinal veins prior to visible nerve changes.

I believe thoroughly in early operation in choked disk, and believe also that if there is doubt as to whether or not the lesion is of luetic origin that the Wassermann test should be used. While this reaction is not always to be depended on, still it is the best means of diagnosis in questionable cases. If there is a definite history of specific infection, especially if comparatively recent, and in the presence of brain symptoms, then I believe a course of mercury should be tried. It is little short of criminal to waste time in every tumor case by such treatment, especially when there is great swelling of the disk and perhaps beginning atrophy. In an old syphilitic case, however, I think the advice of Horsley, to treat it like any other surgical case, is correct. The cases in which operation has seemed to increase the papilledema seem to me too few to weigh much against operative procedure at as early a date as possible; yet, it is wise to inform the patient or friends of possibility of danger.

A symptom, which several of my patients exhibited, I have not noticed reported by others, especially in tumors of the cerebellum and angle; that is, a loss of sense of distance and direction for objects. For instance, in Case 1, the patient said that she missed articles she attempted to reach; either she did not reach far enough, or went to one side, or overreached; and perhaps she would fall over the object. In walking to a door she was likely to miss the opening and strike the casing; and this was not due to staggering, but to a loss of ability to judge the distance and direction of the object.

In Case 3, the patient had noticed the same thing, and stated that she had this trouble, especially in getting into a wagon, and attempting to take hold of the dashboard to aid in getting in; she said that she was likely to miss it and fall, and, in fact had fallen several times.

Another of my patients said that he was likely to stumble over objects that were low, but if the object were sufficiently high he had no trouble in getting over other than staggering; he constantly misjudged the height of objects on the floor, or on the sidewalk, and was likely to stumble over them. This is perhaps but a part of the general ataxic muscular condition.

In two of my patients an early sensory symptom noticed was a feeling of numbness about the eyes and across the face, as if the part were "asleep," and the patient had a desire to rub it.

That brain tumor with rapid degeneration can sometimes simulate brain abscess is, I think, well illustrated in Case 5, as there was the rapid onset, the slow pulse and the subnormal temperature seen in some cases of abscess. I think that in any case in which there are not decided blood-changes and in which no focus of suppuration can be found in any part of the body from which there might be metastasis that we are warranted in excluding brain abscess.

It seems to me that in the last case reported Jacksonian epilepsy is unusual in tumor of the left hemisphere of the cerebellum.

Perhaps my report of these cases, as well as these remarks, is too general in character for the Section on Ophthalmology; but it is my contention that we should embrace in our examination, especially in the absence of a neurologist, a general examination; I have not tried to give here the results of such examination entirely, but only the more prominent general symptoms.

CASE 1.—*Patient.*—R. B., girl, aged 22, teacher, was referred to me by Dr. Colegrove in September, 1897. She looked well, except that she was somewhat anemic, but told me that for a year past she had been in poor health. She had been treated for indigestion, and had also some pelvic trouble. Recently her eyes had not felt right; they smarted and vision blurred, especially in the right eye. At times there was temporary loss of sight in both eyes, lasting from a few seconds to ten minutes, during which time she was very dizzy, and the blindness was seemingly equal in both eyes.

*Examination.*—Vision in the right eye was 20/50, in the left, 20/30. The right disk was best seen with plus 3. There was 1 D. hyperopia, and left papilledema of plus 1. There was a macular change in the right eye, with some chorioiditis, and small hemorrhages throughout the right retina. There was some loss of station, and the patient staggered in walking, chiefly to the right side. She said that she was always running into articles of furniture about the room; frequently made a misstep in descending stairs; that she misjudged distances, and in reaching for an article overreached it, and was likely to fall. Patellar reflexes were lost. Pupils contracted to light and convergence; there was no paralysis of extrinsic muscles, and no nystagmus. The patient has had very severe headaches with severe vomiting, which nothing would control. She said she had taken on some weight within the last few months, but that it did not seem natural to her, and she could not account for it.

*Progress of Case.*—Three weeks later, the right disk had 6 D. of swelling, and the left 1 D. There was much loss of coordination; and more hemorrhages in the right retina. The arteries were small, and the veins dark, large and tortuous; the left retina had a slight macular change. Vision at this time was, right 20/50, and left 20/30.

The patient was put on iodid and mercury, although there was no luetic history.

November 20: The patient said that the vomiting was better, the attack being less frequent and not so severe, but that she still had the blind spells, and that she did not walk any better. She had noticed also that there was loss of taste, and that things did not appear natural to her when eating. There was also some loss of smell; she said that she could not detect the odor of flowers. She appeared weak and anxious. I informed the family of the gravity of the case, and asked them to consult a cranial surgeon; but at this time we did not have as much to offer in this class of surgical cases, consequently I did not urge the matter.

About two months later I saw this patient again. There was 5 D. of swelling in the right eye, and 1 D. in the left, with beginning atrophy to the temporal side of both disks, and the swelling subsiding. There was very severe vomiting at times, her other symptoms were exaggerated, and she had begun to lose flesh.

*Termination of Case.*—I did not see the patient again, but learned from her physician that total blindness ensued; there was almost constant vomiting, inability to walk or stand, and death took place in April. An autopsy was refused.

CASE 2.—*Patient.*—H. S., aged 18, farmer. Family history was good. I saw him in December, 1903. About six months previously he had begun to have severe pains at the vertex of the skull, and became very dizzy, staggering when he walked. He often had attacks of vomiting without preceding nausea. He was dull mentally, and was very slow in his answers; said he was unable to think clearly, and that his memory was poor. Recently his eyes had blurred, and there was a temporary loss of sight in both eyes, varying in time from a few seconds to several minutes.

*Examination.*—He was brought to me with a diagnosis of locomotor ataxia. The patient was very ataxic, chiefly to the right side; there was swaying, chiefly to the right, on standing with his eyes closed and feet approximate. The patellar reflexes were lost. Pupils reacted to light and convergence; there was no paralysis of extrinsic muscles. Vision, right 20/40, left 20/200. The right fundus showed choked disk, best seen with plus 6; left, plus 4. There was no nystagmus, and eye movements were carried out normally; there were no hemorrhages in either retina. On a rough test, both fields were contracted, but color fields were not taken. Some albumin was found in the urine, and some casts, chiefly of the hyaline variety. The patient had become very fleshy in the past few months; the skin seemed tense and hard, as if fully distended; did not pit on pressure, and had a leathery feeling. There was no deafness in either ear, and there was no loss of taste or smell.

*Course of Disease.*—I saw this patient but once, and on telling the family the nature of the disease other physicians were consulted, who, after examination, concluded that it was a case of myxedema, owing to the rapid taking on of fat, the condition of the skin, the albumin present, and the eye-ground condition. These physicians informed me that the man seemed to improve on thyroid extract. He then consulted Dr. Allen Starr, of New York, who diagnosed the case as one of brain tumor. His condition gradually grew worse; his vision failed utterly; moving about, walking or standing without support was impossible; vomiting and headache became very severe, and the patient died suddenly, about six months after I first saw him.

*Necropsy.*—The brain and kidneys were sent to me for examination. They were hardened in formaldehyd solution. The kidneys showed some cortical nephritis. The cerebrum was normal throughout, except that on opening the lateral ventricles there was a thick fluid, slightly tinged with blood in both lateral ventricles. This was spread out on the floor of the ventricles. On section of the cerebellum the right hemisphere was seen to be the site of a large tumor, which occupied the entire side and extended to the left hemisphere but did not involve all of that side. The mass was well broken down at the center, and over the fourth ventricle, and the fluid spoken of as being in the lateral ventricle was in the fourth also, and evidently came from this softened vascular tissue. Examination showed the tumor to be sarcoma.

CASE 3.—*Patient.*—C. F., female, aged 32, a clerk, was first seen in December, 1904. Two years before, she began to have a queer sense as of drawing or numbness across the face, below the eyes, and about the eyebrows. She said that the stiffness annoyed her greatly; she wanted to rub her face to make it feel alive; she also noted at this time that vision blurred greatly. She was examined by an ophthalmologist, whose notes show no disease of the eyes; no macular change; and no refractive trouble requiring glasses. There was, however, 3 degrees exophoria. The numbness persisted, however, and she noticed that she stumbled over objects, and had dizzy spells. She was more apt to stumble over an object if it were low than if it were standing at an ordinary height. She said that she also seemed unable to avoid articles of furniture about the room, or at the store where she worked. If she put her hand out at times to touch an object she was likely to miss it, or to place her hand beyond it and run against it or fall over it. She noticed also that sometimes she made a misstep, and had noticed for months that in approaching customers in attempting to stop she frequently bumped against them, and several times had pitched forward, falling in several instances against the person she was approaching.

She again consulted an ophthalmologist about a year after the first one. This physician made a diagnosis of albuminuric retinitis, as there was some neuritis and macular change. At times also there was a small quantity of albumin in the urine, but no casts. This neuritis and macular change was no doubt the beginning of the papilledema. For some time also, even before consulting an ophthalmologist, she had what she termed "bilious spells"; she would waken suddenly in the night with severe vomiting, expulsive in character, and which nothing would control. She had temporary loss of vision, sometimes lasting for a minute or two, sometimes very transient. She told me she had noticed that for at least two years, in getting into a wagon, she would reach for the dashboard to support herself and would miss it, and had fallen in doing so several times and had become afraid to make the attempt, either to get out of or into a wagon without help, and that her legs seemed weak. Her mother had noticed also that she staggered in walking; that she was continually bumping into things about the house, and had fallen over a chair several times. Vision had rapidly failed, and her vomiting attacks grew so frequent and she staggered so badly that she gave up her position and remained at home. Her ophthalmologist said that nothing could be done for her; that the kidney disease had made her lose her sight.

*Examination.*—When I saw her there was no vision in either eye, nor had there been for six weeks, when she said the last light perception failed. The pupils were widely dilated with no reaction to light; the right cornea was anesthetic, the left partly so. There was marked exophthalmos of both eyes. There was still some evidence of papilledema; both nerve-heads were atrophic. I was unable to decide in which eye there had been the first or greater edema, as both nerve-heads seemed equally atrophic. In both retinas there were remains of numerous hemorrhages, which were nearly absorbed. I could not make out any macular change. The veins were large, dark and tortuous, and the arteries small, with deposit along their sides near the disk. There was marked nystagmus of both eyes, much exaggerated on extreme rotation to the right; the left eye turned to the outer side a little. There was very little headache, but what she did have was occipital. The patient had difficulty in speaking; there was no paralysis of the vocal cords, and the tongue protruded straight, but the left masseter was weak. There was total loss of taste and smell, and there was partial deafness in left ear, both for air and bone conduction. There was a small amount of albumin in the urine, but no casts. Patellar reflexes were lost; the patient was unable to stand with feet approximate, but could do so with feet apart, though with great swaying; she staggered badly on attempting to walk, chiefly to left side. In fact, she would fall after she had taken a few steps unless supported.

*Course of Disease.*—A Christian Science healer stepped in at this juncture of the case, and after receiving glittering promise of cure the easily deluded family asked me to step out, and let her try. After several weeks, and after the healer had assimilated the family's store of funds, which were very limited, I was again called in; but did not claim ability to cure, as did the healer. I advised operation, but it was refused, as I could not, of course, promise return of vision. The girl finally lost all ability to stand, was absolutely deaf in the left ear; had great difficulty in swallowing anything, but could swallow semi-fluid better than fluid. She was unable to converse so that any one could understand her but her mother. There was a constant flow of saliva, and the left corner of the mouth drooped. The patellar and plantar reflexes were abolished. The optic nerves were completely atrophied, and no light sense remained. The patient continued for some months in this condition and finally died suddenly.

*Necropsy.*—There was no change in the cerebrum or ventricles, but there was a large pear-shaped tumor in the left cerebellopontile angle, which had attacked the eighth nerve, and this nerve was embedded in its substance. The growth was also adherent to the periosteum of the petrous portion of the temporal bone. The pathologist's report follows:

"Specimen consists of the tumor of the brain measuring 4 cm. in its greatest diameter. The tumor mass seems to be well encapsulated and to have grown equally in all directions. The cut surface presents a mottled, dirty gray, reddish and whitish coloration, the red areas being due to numerous blood-vessels distended with blood. The white, apparently fibrous tissue strands, have a more or less radiating distribution. The consistency is variable in different areas, being greater in the dense, white connective tissue strands.

"Microscopic examination reveals an arrangement of the cells, diagnostic of fibrous sarcoma. The sarcomatous elements do not predominate, and the tumor histologically seems not to be very malignant, but the size of the nuclei is irregular. The peculiar arrangement of the cells and of the blood-vessel formation, positively indicates a malignant condition of the fibrous tumor. The adjacent brain tissue, beyond some softening, shows no important changes."

CASE 4.—*Patient.*—E. D., female, aged 24, a domestic, was first seen in January, 1906. She had been told, from an examination of her eyes and the presence of albumin in the urine, that she had Bright's disease. About two years ago she noticed that her legs were weak; she staggered in walking, but did not fall. For a year past she had thought her eyes were not right; she could not see clearly, and frequently everything before her eyes would disappear; she could not see for a few seconds, then things became clear. She began to have nausea and vomiting, and about this time noticed that she was growing deaf in the right ear. For three months before I saw her she had severe headache.

*Examination.*—She was blind in each eye, but light perception was present. The ocular movements were carried out in a jerky manner, and I thought that I could detect nystagmus at times, although this was transitory, and not exaggerated, in either direction. Pupils were dilated; the right pupil responded when light was thrown in from the temporal side, in which position it was recognized. The left pupil did not respond to light. There was choked disk in each eye and also advanced atrophy in each disk; there was 6 diopters swelling right and 7 left. There was no paralysis of the ocular muscles. In the right ear there was hearing only for the Galton whistle; bone conduction was nearly lost. In the left ear hearing was not impaired. It was impossible for the patient to walk or stand without being supported; she complained of a queer feeling as of numbness in the right side of the face. The tongue was protruded straight; and the right eye-ball seemed a trifle more prominent than the left. The patellar reflexes were a little exaggerated, but no Babinski sign could be elicited.

*Operation.*—I told both the patient and her family of her condition, and referred her to Dr. Charles H. Frazier, of the University of Pennsylvania Hospital, who, in conjunction with Dr. de Schweinitz and Dr. Spiller, examined her, and concluded that an operation was necessary. On February 23, Dr. Frazier removed the bone overlying the right cerebellar hemisphere. Following the operation, and before the next one was done, the choked disk subsided from 7 to 3 diopters; but of course there was no return of vision. The hearing improved, and the nausea and vomiting ceased; headache disappeared, and there was improvement in ability to walk, so she could get about the ward with very little assistance.

*Subsequent History.*—On April 7, owing to the fact that headache and vomiting had recurred, it was decided to make an attempt to remove the tumor which was supposed to exist. On opening the dura, the cerebellar hemisphere was found to be under considerable tension; it was almost impossible to expose the cerebellopontile angle, but on introduction of the finger a tumor about the size of a hen's egg, firmly adherent in the angle between the pons and the cerebellum, was discovered. The tumor was removed almost intact, and proved to be fibrous.

For the first few days after the operation, while the patient suffered considerable from shock, yet her condition was not such as to cause any apprehension. However, about the sixth day after the second operation, the circulatory and respiratory apparatus began to fail, and the patient died[1] April 15.

CASE 5.—*Patient.*—J. L., aged 25, female, consulted me on Aug. 31, 1906. She came to me because of severe headache, and said that at times she had loss of vision, lasting from an instant to several minutes. She also said that at times she had some nausea. She said for at least six years she had been a sufferer from headaches, the attacks varying greatly in intensity, but for the last six months they had been much more severe than ever before. Her sister, who was with her, did most of the talking; and until I requested her to let the patient answer my questions the patient herself had volunteered little information.

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[1] A more complete general report by Dr. Frazier of this case can be found in THE JOURNAL A. M. A., Sept. 22, 1906, p. 923.

*Examination.*—Vision in each eye was 20/15; under a mydriatic it was the same. The patient was very slow to answer questions; and it seemed to be a long time before she comprehended the meaning of my questions. She was apathetic, seemed confused in manner, and said also that she had been depressed recently. The right disk had beginning neuritis and swelling, chiefly under the superior and inferior retinal vessels as they emerged from the nerve-head. This swelling amounted to 3 D. in the right eye. It shaded off rather abruptly, and the rest of the disk margin on both sides could be plainly seen, although there was some redness all over the disk. The same change existed in the left eye, where the swelling was at the superior and inferior disk margin; the vessels overlay the edema. The swelling was measured with plus 2. The disk margins were plainly seen, both to temporal and nasal side, except where the swelling had taken place, as stated. In the left eye, there was a very little transudation to the outer side of the disk, where a vein crossed an artery. There was not much, if any, change in the size or color of the vessels. I could not make out that there was evidence of venous obstruction. The field, roughly taken, seemed but little contracted, if at all. I did not try the color field. The patellar reflexes were normal. There was no nystagmus, or paralysis of the eye muscles. Plantar reflexes were lost, but station was good. Ear tests were negative, and the finger and nose test was also negative. The grasp of the two hands was equal.

*Course of Disease.*—The next day I saw the patient again, and her condition was the same. I asked her to report again in two days, but she did not do so. However, one week from the day I first saw her I was called to her home. I found her in bed, and only semi-conscious. Both pupils were widely dilated, and did not react to light. Both disks were choked, and each had 7 D. of swelling. There were large hemorrhages in both retinas, and drooping of the right eyelid. Pulse was 50, temperature 98, and respiration 14. The patient did not give evidence of much suffering. She could swallow readily, but I could not get from her intelligent answers.

Three days later, complete ophthalmophlegia took place in the right eye, and paralysis of external and internal rectus of left eye, but the patient could move the left eye up and down a little. Both eye-balls were somewhat prominent. Pulse was 45, temperature 97, and respiration 16. The disks measured about the same in swelling; both pupils were widely dilated, and did not react to light. The patient's answers were very confused as to what she could see. She said at one time that she could count fingers, and again that she could not. She did not seem to notice the bright light from the ophthalmoscope when her eyes were under examination. Patellar reflexes were lost, and no Babinski sign was present. There was no motor paralysis, except of eyes as stated. From the very rapid onset I thought that there must be a focus of pus some place in the body, and again looked over the ears for any source of pus there, but they were normal throughout; still I thought that there must be a brain abscess, because of the slow pulse and subnormal temperature. There was no leukocytosis, and the hemoglobin was nearly normal in amount.

About one week after I first saw the patient in bed she had several convulsions. They usually began in the left arm and were Jacksonian in type, and did not seem to be brought on by change in position. After having had several of these convulsions she seemed to brighten up, and could speak much more clearly, yet remained in a very confused condition. In the absence of her nurse, she got out of bed and walked about the room, but her vision was very much reduced, although she asserted that she could see better with the left eye than with the right. There was no sensory disturbance about the face, nor was there loss of special senses. The pulse remained very slow and full; respirations were slow, and temperature remained subnormal. I strongly advised operation, as I thought that there must be great increase of intracranial pressure; and, in the absence of a cranial surgeon and expert neurologist I advised making a right temporal decompression, which was refused. The patient again grew worse, and died four weeks from the time I first saw her.

*Necropsy.*—The left temporal lobe was found to be the site of a large softened tumor occupying the upper and posterior part of the lobe. It was very soft and vascular, but there were no free hemorrhages in its substance. The rest of the brain was normal, and there was no pathologic fluid in the lateral ventricles. The mass proved to be a sarcoma.

CASE 6.—*Patient.*—M. J., female, aged 51, had always been healthy until about two years ago, when she suffered an attack of influenza, which left her weak. After getting up, she noticed that she was dizzy at times and occasionally vomited. She grew so dizzy after some weeks, and staggered so much in walking, that she used a cane, and had been walking with a cane for over a year. On one occasion she fell during a period of dizziness, and the local physician said that she had fractured two ribs.

*Examination.*—I saw her Oct. 1, 1906. She complained of severe headache, chiefly in the frontal region. She said that she had blind "spells"; and that she was losing her hearing in the left ear; and had very troublesome noises in that ear. Her right ear was normal; with the left ear she thought she could hear a watch when pressed close, but there was no bone conduction. Her vision was, right 20/20, and left 20/20. The pupils reacted to light and convergence; both disks were choked. No hemorrhages could be seen in either eye. The right disk was best seen with plus 6, left with plus 7, but there was 3 D. of hyperopia in each eye. The patient was ataxic, pitching chiefly to the left side, and was apt to strike against the side of the door in passing through. She swayed badly with the eyes closed, chiefly to the left side. The right patellar reflex was retarded; the left was lost. There was some impairment of smell and taste. The Babinski sign was not present. The patient had consulted several ophthalmologists, and had been told she had spinal trouble. One ophthalmologist told her she probably had basilar meningitis. On examination, the urine was found normal.

*Operation.*—I referred the patient to Dr. Charles H. Frazier, who did a decompressive operation in the left occipital region on October 15. A cyst was found underneath the left cerebellar hemisphere and was evacuated. On retracting the cerebellum, a tumor was found in the left cerebellopontile angle; it was very adherent to the surrounding structures, but was not removed. A small section was taken, however, and examination showed it to be a fibrosarcoma. Following the operation, the patient did nicely, but about two weeks later began to have severe nausea, vomiting, and considerable pain. A second operation was done, and the intracranial pressure was found to be considerable; the dura was slit freely, and the wound closed. For some days the vomiting seemed to be increased. She was very ataxic. She also developed some rise of temperature, which ranged from 99 to 102 F. This soon cleared up, but the vomiting persisted.

*Subsequent History.*—When the patient was sent back to me on January 7, the right disk measured 2 D., and her left 1 D. She said that she still had periods of vomiting, and staggered in walking; and had some difficulty in swallowing. Vision was better, and her sense of taste and smell had partly returned.

July, 1907: She had but 1 D. of swelling of the disk in the right eye, with vision of 20/20; and congestion only of the disk in the left eye, with vision of 20/15. With her reading glasses she reads J 2. I thought I could detect some beginning atrophy. She still walked with a cane, and said that she did not remember well.

June 16, 1908: The swelling of both disks had disappeared, but there was evidence of former choking, and some deposit along the vessels near the disk, although the vessels were not altered in size. The patient could hear a 512 C fork in left ear for twenty seconds, but had no hearing for watch on contact. She complained of some numbness in her left hand and arm; and that she frequently choked in swallowing liquids.

Feb. 26, 1911: The last time I saw the patient, there was no swelling of either disk, but still some redness, and showed deposit of fibrous tissue about vessels. With her distance correction she sees 20/15 in each eye. She has a little bone conduction in the left ear, but there was no hearing for watch. She has been going about her duties since I last saw her; she works about her farm, but says that she has noticed that frequently she drops things from the left hand, and that her gait has been very unsteady for the last year, and she feels much more confidence in herself if she carries a cane. There is a large hernia cerebri about the size of a small hen's egg at the site of the operation. She says that at times this is much harder, as she expresses it; and that at those times she notices she is apt to be less steady while walking. She still has some trouble about eating; says that liquids no longer choke her, but that solids frequently do. She suffers no pain; is satisfied now that she will keep her vision; is cheerful, and regrets that she did not have her operation earlier.

CASE 7.—*Patient.*—J. D. M., male, aged 23, machinist, consulted me March 25, 1908, for severe headaches, which he stated he had had since boyhood. Recently they have been much more severe; and about three weeks before consulting me he began to have attacks of vomiting. The vomitus, he stated, was bilious. He has not been free from headaches for three weeks, and the pain is referred to brow and occiput. Just before these severe headaches came on he had noticed dimness of vision at times, and occasionally diplopia. The latter was not constant, however, and was more apt to come on when he was tired. There was no nausea preceding the vomiting. He has vertigo, which began about six weeks before I saw him, and he has had dizzy spells daily since. On two occasions he had fallen, when these dizzy spells began, once on the street. After this attack he seemed confused mentally and did not appear well. With the vertigo he says he feels a peculiar sensation in his left hand and arm, like the arm being "sleepy." Patient had not been working for the past six weeks. He seemed slow of comprehension in answering questions.

*Examination.*—Vision in right eye was 20/15. Patellar reflexes were normal. The patient had a homonymous diplopia. The right disk had 3 D. of swelling, and the left 2 D. The right ear was normal for watch and bone conduction; in the left ear watch could be heard but 10 inches, and whisper 5 feet. I told him of the nature of his trouble. He had denied any history of lues; and on close questioning there seemed to be no specific family history.

*Consultation.*—I referred him to Dr. Frazier, of Philadelphia, who, in conjunction with Dr. Spiller and Dr. de Schweinitz, examined him. Their opinion was that he probably had a brain tumor, but that an operation was not imperative at that time. It was also suggested that he might with safety wait a little time while a course of mercury and iodid was tried, which was done.

*Course of Disease.*—The patient soon began to improve; his vertigo and vomiting subsided, the neuritis rapidly lessened; and on examination in the following June he had but 2 D. of swelling in the right eye and 1 D. in the left, chiefly to the upper side of each disk. He complained of some headache, and of deafness in the left ear. He was feeling very much better in every way, was quicker of comprehension; had returned to work, and had not fallen or noticed that he staggered recently. I have examined him from time to time since; and on my last examination, Feb. 26, 1911, Dr. Frazier being at my office, there was no swelling of either disk, but both were reddened. The patient said that he had worked steadily, and had splendid health since being given the specific treatment; and that he had kept it up at times ever since his leaving the hospital. There is no diplopia, and the patient feels that he is cured.

Case 8.—*Patient.*—In June, 1908, I was on my vacation, and was asked by a physician in a small country town to see what he thought was a very obscure case in a child of 2½ years. The child had been ill for several months; had had difficulty in retaining food, had not been able to walk, and an attempt usually brought a fall, more often to the left side.

*Examination.*—I found the child sitting on the floor, its left hand at its side, spread out on the floor as if in the position of bracing itself. The head was large and undoubtedly hydrocephalic. The child looked fairly well nourished. There was no dilatation of the pupils, and the reaction to light was normal. There was no strabismus. Having no ophthalmoscope with me, and feeling sure that the trouble was central, as the attending physician had suggested, I left some atropin to be instilled; and the next day I again visited the patient with a retinoscope of my own devising—a small toilet mirror, from which I had scraped some of the silvering on the back of the mirror—with which I examined the fundi. I found choked disk in each eye, but was unable to estimate the amount of swelling.

The case occurred some miles from Fort Wayne, and I referred the patient to Dr. A. A. Bulson, who wrote me, under date of July 8, 1908, as follows: "The child has double choked disk, and all the elements of intracranial pressure; is unable to walk or sit up or use its arms. The tendon reflexes are entirely absent. There was but little response to the pupillary reaction, and the family reported some transient facial paralysis, and while the child was in Fort Wayne it developed a partial facial paralysis on one side. Owing to the unfavorable outlook given the family, they took the child to an osteopath, who diagnosed a vertebral dislocation, which he said he could cure. I do not doubt this was a cerebellar lesion, although there seemed to be a general meningeal involvement."

The child is still living but is totally blind; it can recognize different members of the family by their voices. The child has occasional convulsions, is helpless and is only able to utter a very few words. The head is extremely large.

CASE 9.—*Patient.*—In February, 1908, I was called by Dr. A. W. Booth to see C. Y., aged 55, an accountant. For some months past he had had what he supposed was digestive trouble, and had frequent attacks of vomiting. He also had severe attacks of vertigo, with dim vision lasting from a few seconds to several minutes, at which times he said he had great difficulty in maintaining his balance. He had also noticed for some months past that in walking he invariably deviated to the left; and it was difficult for him to follow an ordinary walk without unconsciously getting to the left side. He had also noticed that at times his legs seemed weak, and that he staggered. The vomiting was expulsive in character, and at times he choked while eating. The vertigo and vomiting had necessitated his going to bed, and if he was lying prone he was much more comfortable; only that at times he had severe vertigo in this position.

*Examination.*—The patellar reflexes were normal. The field for form roughly taken was normal. There was no paralysis of eye muscles, nor was there drooping of the lids. The movements of the eyes were carried out in a somewhat jerky manner, and the patient was unable to rotate either eye to the left side as far as he could to the right. At times he had some nystagmus when the eyes were rotated to the left. There was no defect in vision in the right eye, and there were no fundus changes. In the left eye, the upper margin of the disk was a little obscure, and there was a little congestion of this disk, but not what one would call a neuritis; vision was normal. Sitting up quickly in bed was apt to bring on vomiting and an increase of nystagmus. The patient had no loss of taste or smell, but was totally deaf in the left ear.

Three years before he had consulted me concerning his ears; and my notes show his bone conduction in that ear at that time was not normal; and his hearing for the watch 10 inches, and he suffered from tinnitis aurium. At this time he did not complain to me of any of the symptoms here given; and thought that inflation several times did his hearing good. He, however, did not return to me for further treatment.

If he was set upright in bed, and especially if taken out of the bed onto a chair, he began to have attacks of typical Jacksonian epilepsy. The attack began invariably in the left hand and arm; then spread to the left foot, and rapidly became general in character, and was of short duration. There was no exaggeration or loss of patellar reflexes. The patient was clear in mind, but was often puzzled to speak the word he wished, and at times his speech was difficult. He had no sensory disturbance or motor paralysis. From his inability to retain food he rapidly emaciated, and died about six weeks after I first saw him.

*Necropsy.*—The cerebrum was found normal throughout, but the left hemisphere of the cerebellum to the outer side was the site of a large, irregular, softened mass, which on examination was found to be sarcoma.

408 North Main Street.

## MEMORANDA

# INTERSTITIAL KERATITIS OF TUBERCULOUS ORIGIN

## WITH SPECIAL REFERENCE TO TREATMENT WITH TUBERCULIN *

ROBERT SCOTT LAMB, M.D.
WASHINGTON, D. C.

It is now twenty years since Koch's tuberculin, known to-day as old tuberculin (O. T.), came into use. The results which followed were as variable as are those now being reported following the use of salvarsan, and for the same reason, the most unreasonable application in cases diagnosed, often by "snap-shot," as tuberculosis, general or local. Furthermore, regardless of the stage of the disease, the age or general condition of the patient, the dose was the same. And, whereas this state of things might be in part excusable because of the desire to help, by any means at hand, such unfortunate persons, it was nevertheless most deplorable that the use of tuberculin was not attended with greater wisdom, so that the proper cases could have been treated by proper methods, and this very valuable remedy have been retained in our armamentarium during the following ten years. However that may have been, the careful work of men belonging to the minority of workers in medicine, including illustrious ophthalmologists, has during the past ten years put this remedy on a firm foundation. Tuberculin has been found useful in some cases and only partially useful in others, but the consensus of opinion now is that in one or another of its forms or preparations it is of distinct value.

In the early years of my practice, I learned that many cases of interstitial keratitis would not respond to anti-

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syphilitic remedies, but would respond to phosphatic emulsion, tonic and dietetic treatment, and this was especially true of scrofulous patients. I came to conclude that the so-called tendency to consumption was the underlying cause of the disease in many cases. Tuberculin had been tried (?), and found wanting.

As I do not wish to take up time with a long account of the use of this drug in its different forms, for that has been done by more than one able worker, even in the past year—Dor, Derby and others—it will be sufficient to point the way to the more frequent use of tuberculin in those conditions in which the usual remedies have failed.

Cases of interstitial keratitis occurring bilaterally in scrofulous patients are undoubtedly the result of mixed infection, both syphilitic and tuberculous, and treatment for either alone will not suffice; measures must be directed to the cure of both diseases by proceeding against either one at a time or both together. Therefore, in addition to the use of sodium or potassium iodid, mercury frictions and salvarsan, one must use cod-liver oil, phosphatic emulsion, diet, tonic, out-door life and tuberculin in well-selected cases.

I would differ with some authorities regarding the advisability of giving such a remedy as tuberculin to out-patients, and would insist on its being given only to those patients who could be taken into the hospital and carefully observed while under treatment. This has been my practice for five years and results have been more satisfactory because of the regimen. Two such patients, so blind that it was necessary for friends to lead them to the hospital, were discharged at the end of six months and thirteen months respectively, with vision in the first case normal, and in the second patient, who had also kyphoscoliosis, vision of 20/40. This patient has been at work now over four years. The first patient, a woman, has married since, and has had one child, which I have seen and which seems to be fairly healthy.

When we consider interstitial keratitis from a micropathologic standpoint, whether tuberculous in character or not, authorities are agreed as to there being few, if any, characteristics typical of tuberculosis, syphilis or injury.

## ETIOLOGY AND CHARACTERISTICS

Any toxic inflammation of the uveal tract may, and often does, have as a complication an interstitial keratitis. It is a question how often the keratitis is primary. Knowing these facts, we must direct our treatment toward the increase of the resistance by general tonics. diet and regimen and the increase in the number of antibodies by direct stimulation of the cells producing them, with a specific or paraspecific serum.

The appearance of this condition is familiar to most of us. The cornea is rather uniformly hazy in the more acute stage, gradually clearing under treatment rather irregularly, which is not usually the case in syphilitic interstitial keratitis. Simultaneous with the rather dense cloudy condition of the cornea, in the acute stage, is the marked deposit of large aggregations of leukocytes in isolated masses with a marked reticulated arrangement in each, on its posterior layers. Of course it is not like the appearance of a deep keratitis following an iridocyclitis in which the masses are rather cheesy, as found in syphilis unassociated with tuberculosis. One of the diagnostic features of such cases is a negative Wassermann or Noguchi reaction, and a positive reaction with the Moro or von Pirquet test. Many of my patients have had this typical appearance above suggested and the reactions with tuberculin were positive with a negative Wassermann reaction.

With the Moro or von Pirquet tests the local and focal reactions have been good, yet there has been little general reaction, whereas with the same patients, 1/500 mg. O. T. has given a very positive general reaction as well as local and focal manifestations. In one case which comes to mind, I recall a marked increase of exudate in the anterior chamber deposited in a ring-like arrangement with a clear center opposite the pupil, following such a small injection of O. T. as 1/500 mg.

### CONCLUSIONS

So far as the efficacy of tuberculin in its different forms is concerned in effecting cures, I may say that authors, as usual, differ, but from my own experience I cannot say that O. T. is the most beneficial. I did not check the injection with opsonic index findings because of the unwarranted labor and time consumption, but clinically I seemed to see that the reactions, both focal and general, were too great; I was surprised, however, to see that the Moro and von Pirquet tests, although used for diagnosis only, really affected the focal condition very favorably, and so I continued to use the Moro technic in some cases every other night and in others the von Pirquet, also every other night.

Though not prepared to prove it, I feel that the good effect was due to the small, but frequent, stimulation of the antibody-producing elements of the system.

I regret my inability to present the case reports on which the above conclusions are based, having written this article as a preliminary note to a more exact study of these and other cases yet to come. My main desire was, and is, to get other ophthalmologists to treat these cases of interstitial keratitis which do not respond to the usually accepted treatment, using one of these easily applied preparations of tuberculin and thus obtaining earlier and more certain cures in more of these cases, by stimulating those elements of the system whose function it is to produce antibodies.

It may be advantageous to emphasize the following points:

1. Many patients with interstitial keratitis formerly treated in a routine manner as syphilitic and not recovering completely, might have been greatly benefited by the recognition of tuberculosis as a partial cause, or the only cause of the diseased condition of the cornea.

2. Such cases are now more easily diagnosed because of the tuberculin tests and their successful treatment made more certain by the use of tuberculin in one or another of its forms. The particular form selected by the ophthalmologist should be that one which gives the best result with the least physical discomfort.

3. Acknowledging these observations to be true, the application of known remedial agents to the best of our ability may result in the complete cure of some of these cases of interstitial keratitis of tuberculous origin.

## MEMORANDA

# MEMORANDA

# TUBERCULOUS CYCLITIS FOLLOWING NON-PERFORATING INJURY TO THE EYE*

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**Surgeon to Illinois Charitable Eye and Ear Infirmary and Ophthalmologist to Augustana Hospital**
AND
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**Pathologist and Assistant Surgeon to Illinois Charitable Eye and Ear Infirmary**
CHICAGO

## CLINICAL OBSERVATIONS BY DR. DODD

The following case presented some very interesting features. Two of the most unusual conditions were the rupture of the retina, which was present in the region of the ora serrata as the result of a contusion, and an anterior tuberculous uveitis manifesting itself several months after the injury, with negative results from the diagnostic use of tuberculin.

*History.*—The patient, T. L., aged 10 years, was first seen by me in March, 1908, and the following history obtained: The family had noticed a spot on the left cornea for about eight weeks, and although the eye at times was inflamed and at other times free from irritation, this spot remained. The child had been struck over the left eye with a baseball about ten months before; the lids became discolored and the eye-ball inflamed. A few months prior to that he had been struck near the eye with a stone, the injury producing severe epistaxis and great swelling and discoloration of the eyelids. He gave an indefinite history of not having seen so well with that eye as with the right, but could not say whether this reduction of sight followed either one of the injuries or not. His general health was good and no recent illness was reported, although his mother said that at about the time the eye trouble began he complained of headache and was drowsy and slept a great deal, even falling asleep when he came home from school at noon. As he complained of no other symptoms of illness, however, little attention was paid to this. He had had the usual diseases of children, also a suppuration of the ears when about two years old.

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*Family History.*—No history was given of any trouble which could be considered tuberculous or syphilitic in any member of the family. The mother had delicate health as the result of a fall which injured her spine, when she was a small child. The patient had one brother older and two sisters younger than himself, and there was a stillbirth occurring about eighteen months previous to his birth.

*Examination.*—The patient's right eye was perfectly normal, with vision equaling 5/6, but he had not been able to use it in school for some time on account of the sensitiveness of the left eye. With the left eye, he could count fingers at 18 inches. There was some ciliary congestion, which was more marked at the lower part of the eye-ball, and a great deal of photophobia and lacrimation. The cornea had a triangular opacity involving its lower third and extending up to and covering part of the pupil. This was quite dense and the epithelium over this part was stippled as in interstitial keratitis. There were some small opaque spots in the cornea above this, and the pupil was small and did not dilate readily with atropin. The tension was normal, but no view could be had of the fundus on account of the corneal opacity. Atropin and dionin were used in the eye, mercuric inunctions applied, and iodids given internally. Under this treatment the irritation and inflammation of the eye subsided somewhat, and the pupil finally became fully dilated so that examination of the fundus was possible. At the extreme lower part was a whitish mass apparently extending forward to the ciliary body and ending near the meridian of the eye. This was glistening white, nonvascular, and was in front of the retina, no detachment of which could be seen. By the use of the transilluminator I found that light passed through it readily.

*Course of Disease.*—Judging that the corneal trouble, as well as the condition involving the ciliary body, were tuberculous in origin, I used the ophthalmo-tuberculin test in the right eye, but got no reaction. By the latter part of May the eye was slightly improved, the cornea being less opaque and the sensitiveness to light about gone, but on discontinuing the use of atropin the eye would become irritated.

June 22: The patient went to the hospital for a thorough tuberculin test and, after having his temperature taken for several days, I gave him progressive doses of old tuberculin up to the final dose of 10 mg. His temperature remained normal and at no time was there any reaction, local or general.

July 30: By this time, the eye was much improved; there was less photophobia and the cornea was much clearer.

**August 13: He came to see me before going to the country for a vacation. I found the eye quiet with only a slight opacity of the cornea. There was no change to be seen in the whitish exudate, the vitreous was clear, and there was no detachment of the retina, or anything to make one suspect a growing tumor.**

**October 20: He returned on this date, having noticed for two weeks that the sight of this eye was failing. The eye was irritated and would become greatly congested on handling, and the lens was becoming opaque at the lower part. The pupil was fairly well dilated, but the whitish mass could hardly be seen on account of the lenticular opacity. This opacity continued to increase quite rapidly so that by November 9 I could not see into the eye. The iris was pushed forward below apparently as the result of a rupture or dislocation of the lens at its lower part. The eye was greatly irritated. The possibility of an intra-ocular tumor occurred to me at this time, and I requested the family to consult another oculist.**

**November 12: Dr. Suker was consulted and expressed the opinion that a tumor had caused the rupture of the lens capsule and that enucleation should be done at once.**

**November 17: I enucleated the eye, following which the patient made a good recovery.**

***Result of Operation.*—The irritation of the right eye which had been so marked, especially toward the latter part of the trouble, making it impossible for him to use it for reading or school work, subsided as soon as the left eye was removed. I have seen the patient from time to time since and he continues in good health and his vision is normal in the right eye.**

Dr. Lane has made a thorough examination of the enucleated eye and his report of the pathologic findings forms a part of this paper.

When the patient was first seen by me the clinical appearance of the eye was that of beginning interstitial keratitis. A slight diffuse infiltration in the stroma and some roughness and stippling of the corneal epithelium were present, which prevented the principal pathologic condition of the cornea—the endothelogenous membrane covering the posterior surface below—from being demonstrated clinically.

After the dilatation of the pupil and a partial clearing of the cornea the cyclitic mass became visible, and made me consider the history of traumatism of more significance. Later, when the pathologic examination revealed the rupture of the retina and the exudate at its site, the importance of the traumatism as a cause of the trouble was substantiated.

Rupture of the retina rarely occurs without being associated with rupture of the chorioid. When it occurs alone it may be the result of a chorioidal hemorrhage which detaches the retina and finally breaks through into the vitreous chamber, or it may take place from an indirect blow in the same manner as a chorioidal rupture. When this occurs, the most frequent points are at the macula, or at the ora serrata, as in this case. The earlier exudate or organized hemorrhage, having its source at the site of the retinal rupture, was cicatrized and showed no characteristic tuberculous changes. The later tuberculous exudate was from the ciliary body and iris and was still fresh and in a cellular condition.

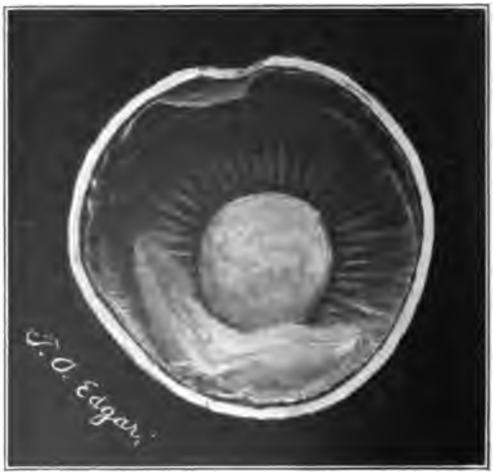

Fig. 1.—Drawing by Dr. T. O. Edgar, of anterior portion of eye, showing mass of exudate.

The long period of time—nine months or more—between the last injury and the time when the inflammation was first noticed is worthy of consideration. Did the infection occur at the time of injury and remain dormant, or did it occur later and become localized at this point of lessened resistance? Either may have happened, for we frequently see cases of tuberculous inflammation which remain quiescent for several months and then suddenly take on new activity due to the lessened resistance of the patient or some local irritation. In one of my patients who had a severe tuberculous sclero-keratitis, healing took place under treatment with tuberculin and the eyes remained well for over two years. On account of working under poor hygienic conditions her general health became affected, she lost weight, and had a recurrence of the same local inflammation for which I had previously treated her. Stock, by his experiments, proved that large tuberculous foci may exist in the ciliary body for a long period without producing any symptoms of cyclitis, and also that tubercle bacilli may be found in the iris months after apparent healing.

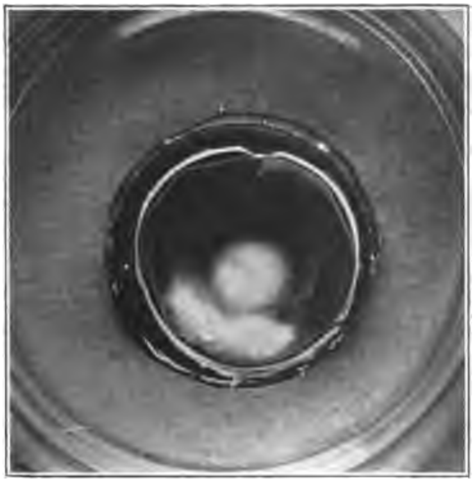

Fig. 2.—Photograph of anterior section of eye.

The absence of reaction to the tuberculin test is very difficult to explain, as in every case of ocular tuberculosis, in my experience, the reaction was well-marked both locally and generally. Not considering the negative ocular tuberculin test as sufficient, I had a thorough subcutaneous test made in the hospital where the patient's temperature could be taken frequently and the condition of the eye noted. Old tuberculin was given in progressive doses until 10 mg. were given, the largest amount I ever use for the test. No reaction was obtained. After learning the pathologic condition of the enucleated eye I again gave him a thorough tuberculin test, with the same negative result. To eliminate hereditary syphilis I had a Wassermann test made and it also was negative.

Localization of the trouble in one segment of the eye is no diagnostic help in the differentiation between intraocular tumors and tuberculosis. The whitish mass which was present in this case might have been a non-pigmented sarcoma, although in so young a patient this would have been very unusual. Solitary tubercle of the chorioid, occurring far forward, is usually of far more rapid growth, and would have produced detachment of the retina visible with the ophthalmoscope at an early date. Tuberculous cyclitis is frequently localized in one section of the eye and may remain so for months before other portions of the eye are affected. The negative tuberculin reaction, however, made me discard the diagnosis of tuberculosis in any of its phases, so that when the extension of the process began again, I concluded that it must be some form of ciliary tumor.

## PATHOLOGIC REPORT BY DR. LANE

### GROSS FINDINGS

*Left Eye.*—Laboratory No. P. 283. (24x23x23 mm.) There is no evidence of a penetrating wound. The entire cornea is superficially hazy, and deep in the lower quadrant is a triangle of fairly dense uniform opacity. The anterior chamber appears a trifle shallow below. The pupil is horizontally oval and measures 5 mm. in its long axis; a thin gray exudate lies in its lower part, and a 1 to 2 mm. broad spindle-shaped opacity, apparently just behind the lens capsule, runs diagonally from axis 60° to 240° through its center.

When sectioned coronally, the anterior half shows the following: The lens is somewhat opaque and is displaced a trifle downward. On the equator of the lower outer third there lies a dense, glistening white, flat exudate, resting principally on the inner surface of the middle and anterior portions of the corresponding part of the ciliary body. The exudate spreads up along the equator of the lens both temporal and nasalward like two wings, the tips of which reach the level of the horizontal meridian. It rapidly converges posteriorly and continues backward and to the temporal side as a short, 3 mm. thick, round stem; its posterior end is attached to the anterior part of the detached retina 1 to 2 mm. behind the ora. The optic nerve is 8 mm. long. The posterior half of the bulb shows no special changes.

Both halves of the eye were fixed in Zenker's solution, imbedded in celloidin, and sectioned parallel to the plane of the meridian passing through the axis 105°. Four hundred and thirty serial sections were made of the anterior half in order to exclude the possibility of a penetrating wound or the presence of a foreign body, and also with the special object of studying the large exudate and the adjacent structures. Eighty-six sections, every

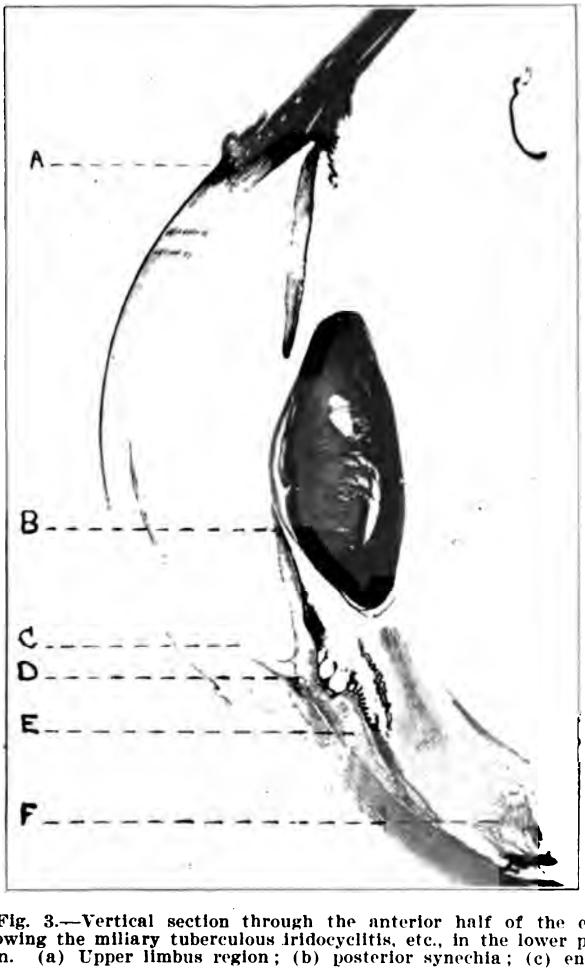

Fig. 3.—Vertical section through the anterior half of the eye, showing the miliary tuberculous iridocyclitis, etc., in the lower portion. (a) Upper limbus region; (b) posterior synechia; (c) endothelogenous cuticular membrane; (d) anterior root synechia; (e) active tuberculous cyclitis; (f) old cicatrized exudate and end of ruptured retina.

fifth one, were stained with hemalaun and eosin, forty-one with carbol-fuchsin, a few by the van Gieson and Mallory methods, and some stained for iron.

### MICROSCOPIC FINDINGS

Unless otherwise stated, the following description has reference to the lower half of the bulb, inasmuch as the most important anatomic changes are found here.

A foreign body is not present. Three distinct, quite superficial, spindle-shaped nodules of round cells are found in front of the perforating vessels in the superficial layers of the sclera (10 to 12, 65 to 70, and 76 to 80)[2] (Scleritis). Files of cells run off from the anterior ciliary vessels and the marginal loops. A few scattered wandering cells are seen, especially between the basement cells of the corneal epithelium, and, moreover, they are increased throughout the interlamellar spaces of the corneal stroma (5).

Near the end of Descemet's membrane the posterior lamellæ are slightly spaced apart and many cells with deeply staining oblong nuclei lie in the narrow fissures (1 and 56). A cellular membrane is found on the posterior surface of the cornea and is worthy of detailed description. It begins at the center, spreads downward and gradually increases in thickness until its superimposed layers reach a maximum of from 12 to 15 strata just above the periphery of Descemet's membrane (1). In some sections this deposit is arranged in particularly definite lamellæ (1, 56 and 57 v. G.), whereas in other sections (10 and 20) lamellation is less regular; here more cells abound, some have large faint-staining oblong nuclei, others have smaller, deeper staining, curved and irregular-shaped nuclei (endothelogenous cuticular deposit). Between Descemet's membrane and this new-formed tissue an interrupted layer of compressed endothelial cells is found. This same arrangement of cells and tissue prevails between the new lamellæ to a lesser extent (1 and 56).

In Section 20, alternating areas are present where the endothelial cells are irregularly grouped, and absent between the new tissue and Descemet's membrane (endothelial degeneration). Between the layers of the deposit a few scattered pigment-bearing cells and free pigment granules can be made out (3) (old precipitates). The cuticular deposit extends a short distance beyond the peripheral margin of Descemet's membrane, overlapping the anterior end of the inner lamellæ of the corneo-scleral trabeculæ (1). Aggregations of leukocytes and plasma cells laden and interspersed with pigment lie in a delicate network of fine fibrin on the posterior surface of the deposit (3) (fresh precipitate).

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[2] Arabic numerals refer to the serial section number.

In Section 35 these precipitates consist almost exclusively of plasma cells. Many lymphocytes and wandering cells of various types are seen in both the angle of the anterior chamber and its bordering walls (1 and 4). In Section 10 an unusual number of eosinophils are present in this location. The canal of Schlemm is completely hidden (6) (partial impermeability of the infiltration angle). Delicate fibrinous strands, in the meshes of which are leukocytes and plasma cells, stretch across the angle (3 and 4) and pull the thickened iris root forward until it is actually approximated to the cuticular

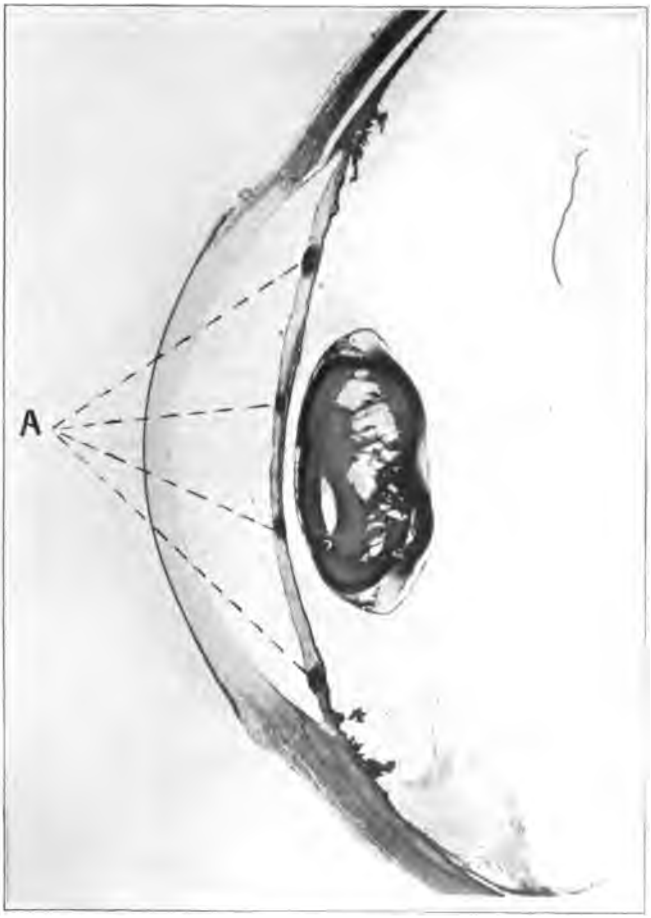

Fig. 4.—Miliary iris tubercles (a).

deposit 1/3 mm. above the end of Descemet's membrane (local peripheral anterior synechia). The canal of Schlemm is here completely blocked. The anterior chamber is free from exudate except at the angle; yet a few plasma cells and leukocytes lie on the anterior surface of the iris, and not a single section shows open stomata of the crypts (cellular exudate on iris surface). The iris stroma, particularly its posterior portion, is thickened by a diffuse infiltration of leukocytes, plasma and mast cells and a few scattered hyaline globules (Russel's bodies) (4). The chromatophores have mostly lost their graceful branches and have become transformed into shapeless conglomerations of pigment. Along with this diffuse infiltration are found many large, fairly sharply circumscribed nodular aggregations of lymphocytes. Several of these nodules reach a size equal to the thickness of the iris and can easily be detected macroscopically (32, 61 and 72) (nodular iritis). Epithelioid cells are present in the center of a few of these nodules (33 and 34), but in none of them can giant cells be found. Vessels are seen inside the nodules in a few sections (32 and 60), yet for the most part they lie at the periphery or entirely outside the nodules. Time was not taken to determine the exact number of nodules present, but their distribution, frequency and characteristics, were closely studied. Fifty-one, or two-thirds of a total of 86 sections studied, show that each section passes through from one to four nodules. In all, 94 cross-sections of such nodules can be counted.

From the observations made one can conclude that (1) the greater number of nodules are located in the lower half of the iris; that (2) they are situated principally in the vicinity of the circulus arteriosus minor; and that (3) they are poor in vessels. These three findings agree with those described in typical miliary tubercles of the iris, and indeed the condition coincides with it entirely except for the fact that giant cells could not be found in a single section.

Attached to the lower pupillary border of the iris is a moderately cell-rich inflammatory exudate. It extends upward for a short distance and tapers off in the pupillary region of the anterior lens capsule (pupillary membrane) and below and behind the pupillary border of the iris and binds the tunic to the lens capsule. This latter portion of the exudate contains a few clumps of pigment (3 and 57). It can be traced downward as a thin pellicle along the posterior surface of the pigment epithelium as far as the ciliary zone of the iris (3) where it suddenly becomes one-half the thickness of the iris (5). The succeeding twelve sections show that the exudate extends, indeed, almost to the ciliary body, and that markedly proliferated pigment epithelium separates it from the posterior surface of the iris; also that it drags narrow bands of pigment toward it and that the exudate fuses with some of the displaced ciliary processes (12 and 13) (posterior synechia). The traction set up by this exudate membrane pulls the pupillary border of the iris backward and downward so that the sphincter is bent on itself in the shape of a hook (3). A thin stratum of connective tissue separates the two somewhat brokendown pigment layers near the outer edge of the pupil (59). The intima of the vessels is often surrounded by small round cells and plasma cells and occasionally a lumen is obliterated (71). The same diffuse cellular infiltration described in the iris prevails in the ciliary body and thickens it propor-

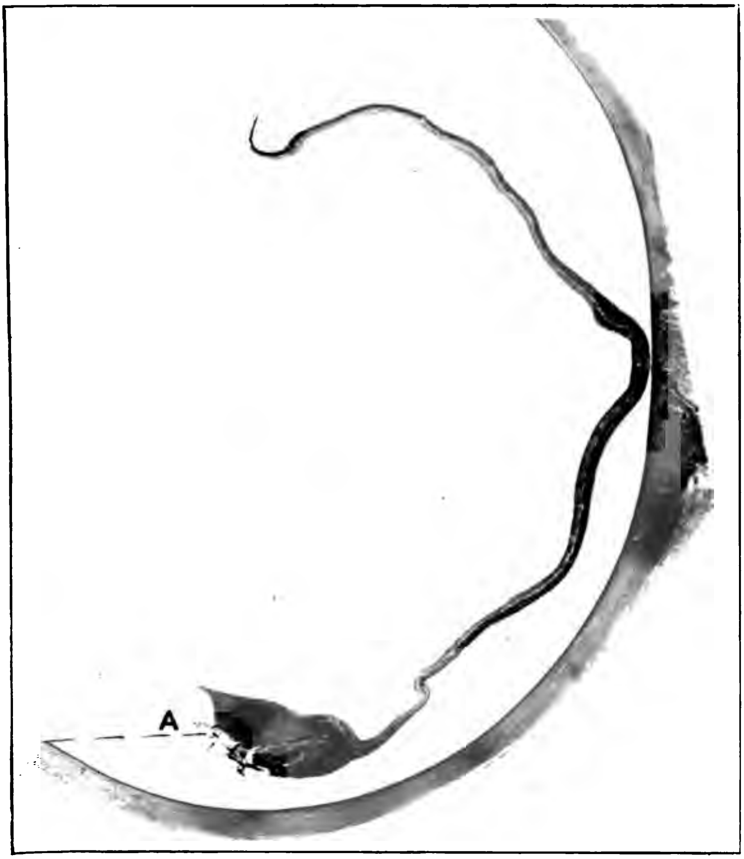

Fig. 5.—Vertical section through the posterior half of the eye, showing the tear in the retina and the old organized connective tissue "callus." (a) Tear in the retina.

tionately. The ciliary processes are most extensively and very densely infiltrated. Some of them are swollen and elongated (8 and 29), others are pushed forward and grown fast to the posterior surface of the iris (26). Still others stretch upward as far as the equator of the lens and lie on the anterior fibers of the zonula (4). In the vascular layer of both the corona (24 to 29) and the orbiculus (36 to 38) a number of round-cell nodules are present. Just behind the corona the pigment epithelium is split and separated from the ciliary epithelium by cicatrical tissue (11 and 27). The ciliary muscle itself has to a large extent escaped infiltration (56). A few sections, particularly No. 6, show the radial bundles widely spaced apart by the same kind of cells that infiltrate the vascular layer, while in others nodules of round cells with numerous eosinophils are found in the immediate neighborhood (24). A diffuse infiltration is present in the lower quadrant (36). Two typical giant-cell systems with central degeneration lie in the muscle opposite the middle and posterior parts of the corona (36 to 40). The stratum pigmentum is broken through in the same sections.

In all, 41 specimens were stained with carbol-fuchsin and a close search made for tubercle bacilli, but bacilli were found in only one section (40c). Morphologically, the bacilli are a little shorter and possibly a trifle thicker than typical tubercle bacilli. A single bacillus having all the morphologic characteristics of the tubercle bacillus was found in smear made from those sections taken from the giant-cell areas and treated by the alkaline - hypochlorite (antiformin) tissue - dissolving method.

A 2 to 3 mm. thick organized exudate lies on the inner surface of the ciliary body and on that (peripheric) portion of the retina which is pulled forward as far as the middle of the orbiculus (2) (cyclitic exudate). It extends upward and imbeds the processes and equatorial zone of the lens, and sends fine filaments up behind the posterior lens capsule, fusing it to the anterior limiting layer of the vitreous. A cellular exudate is pouring out through the broken apices of the swollen processes; it takes a centripetal, axial course (15 to 19) (active exudate). This mass has only a moderate number of vessels (4 and 11), is irregularly infiltrated with the same types of cells found in the ciliary body, and, as in the ciliary body, shows areas where nearly all the cells are eosinophils (15 to 17) (less recent exudate).

While some connective tissue network permeates the more cellular portion, the inner and posterior parts of the exudate are by far the richer in a connective tissue which has become more or less laminated (15 and 27 v. G.) (old organized exudate). Here and there pigment is seen (10). In fifteen of the sections giant cells are to be found (15 and 24). The oldest or most posterior part of the exudate, and the connective tissue which separates the slanting ciliary epithelium from the pig-

**ment layer, lie on the inner surface of the back part of the orbiculus, while the more recent cellular exudate is situated opposite the corona. The mass extends for a short distance behind the ora and is attached to the folded and degenerated, severed retina. These facts furnish conclusive evidence that this mass originated from the region of the ora and not from the corona ciliaris. The lens is slightly subluxated downward. The equatorial zone of the lower half is completely enswathed in the cyclitic "callus" of organized exudate (15) (cataracta accreta). A thin delicate film of connective tissue with a few pigment-bearing cells, and some free pigment, covers the lower part of the anterior capsule. The capsule is wrinkled at the lower equator, probably from hardening. A thin fibrous tissue with spindle-shaped nuclei lies behind the capsule and extends over the entire pupillary surface of the body of the lens. This tissue consists of layers of imbricated spindle-shaped cells packed closely together and does not differ essentially in appearance from ordinary connective tissue (57 and 57 v. G.) (anterior capsular cataract). Fragments of broken-up cortex are imbedded in the capsular cataract. Clefts in the lens substance are filled with fluid and globules of Morgagni (55 and 57) (consecutive cataract).**

**The peripheral part of the chorioid is infiltrated with lymphocytes, leukocytes and plasma cells.**

*Anatomic Diagnosis.*—1. Rupture of the retina following trauma; (2) chronic plastic iridocyclitis limited to lower part and consisting of two stages: (1) An old affection resulting in the formation of cicatricial tissue. (2) A recent affection of the ciliary body which has spread out in all directions, invading the iris and episcleral tissue, evidently along the perforating vessels and characterized by the formation of nodules of round, epithelioid and giant cells with central degeneration—a formation which can be considered miliary tuberculosis.

31 North State Street.

# MEMORANDA

# AN INDEX TO THE GENERAL BLOOD-PRESSURE: THE VENOUS PULSE AND BLANCHING OF THE RETINAL VESSELS INDUCED BY PRESSURE ON THE EYEBALL *

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**DENVER**

By pressure made on the eyeball with the finger placed over the junction of the eyelids at the external canthus, the intra-ocular tension can be materially increased. The tendency of this pressure is to empty the veins of the retina, to inhibit the inflow of blood into the retinal arteries, and to produce the venous pulse.

If, while this pressure is made, one watches the retinal circulation of a young child, the pulsation of the veins on the disk will be first observed; then the arteries will be seen to diminish in size and the veins to be almost emptied of blood. This is because the vessel walls are soft and resilient. In old age the walls of the vessels offer more resistance and scarcely any visible effect from the pressure is observable. In some very elderly people, however, the venous pulse is seen and a decided blanching of the vessels takes place while making steady strong pressure. These are the exceptions and only go to prove that a small amount of vascular hardening has taken place, or that this individual has always enjoyed a normal blood-pressure. It is admitted that this test is not of great value in very elderly people, for the reason that almost all of them will show little or no change in the retinal circulation from pressure. Its greatest value is in young or middle-aged subjects. If, while examining

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the eye-grounds of a patient aged 30, we apply pressure to the eye-ball and find that we cannot cause the venous pulse or blanching of the retinal vessels, we have good reason to suspect increased blood-pressure, and almost invariably the sphygmomanometer will confirm our suspicions. The early recognition of high blood-pressure may mean much to this individual.

A very striking picture is presented in people who have dilated hearts with very low blood-pressure. Here pressure on the globe causes a decided emptying of the arteries, with but little effect on the veins. When arteriosclerosis is well advanced, no matter what the age of the subject, or whether the general blood-pressure be high or low, little or no blanching of the retinal vessels will take place from making pressure on the globe, and it is equally difficult to produce the venous pulse. I have now given the matter close attention for four years, and I find when referring a patient to an internist for examination that I can almost invariably foretell what the blood-pressure findings will be. This does not mean that this pressure test renders the use of the sphygmomanometer unnecessary. On the contrary, it means that the ocular findings warrant a careful estimation of the blood-pressure by the use of the sphygmomanometer.

I have been unable to find in the literature any reference to this pressure test. If any one is before me in having published anything on this subject I shall be only too glad to give him due credit and to apologize for stealing his thunder.

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## MEMORANDA

## MEMORANDA

# SUBHYALOID AND VITREOUS HEMORRHAGES *

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This paper is a study of a subject of which we confessedly know very little, and is undertaken for the following reasons: 1. Its importance. 2. The possibility that what I shall call the "new attitude" of members of this Section toward ocular lesions of uncertain etiology may, during discussion, bring out important truths. At any rate, this "attitude" means that oculists are awakening to their own responsibility of tracing systemic causes, not simply making the diagnosis of ocular condition and referring patient and diagnosis to some physician, who, the oculist is often sure, will go no farther.

This mental change on our own part is worthy of emphasis, and if, for the time being, we can do little more than recognize the problems, we should keep their existence constantly before us. Their nature may be briefly stated: Syphilis, tuberculosis, rheumatism are thought to be the most prolific causes of eye diseases. The first is often undiscoverable clinically or in the history of the patient. The same is true of the second. As to the third, excluding gonorrheal arthritis, and limiting consideration to the disease arthritic rheumatism, the average oculist, after years of experience, can probably count on the fingers of one hand the cases of "rheumatic" eye disease which he has observed. Yet when he enters the great *terra incognita* of "chronic" rheumatism, "muscular" rheumatism, etc., cases abound. He cannot define this condition; neither can his confrère, the internist.

Modern methods of studying mysterious eye lesions are based on the use made of laboratory evidence and pathologic results of syphilis, tuberculosis, and what really underlies that group of general symptoms to which the term "chronic rheumatism" has for years been applied, and which, in one form or another, so often are associated with ocular lesions. How study of this sort has taken us into fields we little dreamed of, one can easily see by looking at our proceedings of the past few years. What is true of the three systemic troubles mentioned, is equally true of constipation, menstrual and pelvic anomalies, anemia, malaria, nephritis, pregnancy, etc. In other words, given an ocular condition, functional or organic, not accounted for by the state of the eye itself, it becomes our duty to note everything wrong anywhere, and to determine the effect of this on the eye.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

It was in this spirit, and at the request of our Chairman, that I undertook the study herein presented. I felt in the beginning that my personal experience and knowledge were totally unequal to the task. A review of even our newest text-books showed that little regarding underlying conditions was known. The objective appearance of the vitreous and of subhyaloid hemorrhage is given. Some authors stop there. Others state that the source of the hemorrhage is usually the uveal circulation, with the retinal vessels occasionally responsible. A few, notably, Fuchs, de Schweinitz and Weeks, tell about the reasons for the break in the vessels. But all this is rather suggestive than conclusive, and necessarily so; for it is not the function of a special author to do more than suggest lines of general investigation. In current literature, many cases are reported with nothing except the objective findings, age of patient, etc. In only a few, so far as I have been able to discover, are the reports specially instructive.

Then I decided to turn to men in our own Section, men of large clinical experience, trained observation and judgment, and to ask their aid. They were all busy men, and for their help I wish to express the warmest thanks. Many took the trouble to analyze their cases, while others, with no time to do this, gave, what is just as valuable, convictions based on long observation. I regret that some of the replies have come in only while this introduction is being written, and too late for incorporation in the report. Nor could I, without making the paper inexcusably long, even seek information from all available sources. A few special features were dwelt on in my inquiries: age, condition of the eye, systemic lesions, blood-pressure, future of the affected eye and of the patient. Some of my correspondents had never seen cases, and one asked what I meant. This is not particularly surprising when one considers that in an article by Scrini and Bourdeaux,[1] it is stated that essential relapsing hemorrhages appear about once or twice in 5,000 cases. Following are some of the replies I received:

### REPORTS OF OBSERVATIONS

**Dr. Melville Black**, Denver: Youngest patient 21, oldest 72, When blood-pressure was low I usually found dilated heart or auto-infection. With high blood-pressure, arteriosclerosis, incipient and advanced kidney degeneration. Blood-pressure was taken in every case in last eight years, and was less than 110 in 60 per cent.

*Ultimate Results:*—Glaucoma resulted and enucleation was done in two cases. Young patients with low pressure usually recovered, though one patient had four relapses. General health normal. Older patients with arteriosclerosis, nephritis, high blood-pressure did well or badly according to progress of general disease. Absolute milk diet recommended for reduction of B-P.

**Dr. James H. Bordley**, Baltimore: *Case* 1.—Female, aged 52, had repeated hemorrhages from 17 to 29; dense hyaloid opacities in right eye, in left, vision = fingers (?). Both lenses, which were clear, were removed and hyaloid needled. Right eye showed proliferating retinitis. Left recovered V. to 10/50.

General condition: Aortic stenosis; blood-pressure 190; nephritis, indicanuria; patient later developed carotid aneurysm.

*Case* 2.—Woman, aged 56, had vitreous hemorrhages from 18 to 30. Sight completely lost at 40. Lenses cloudy, no light perception in either eye. Aneurysm of aorta, nephritis, indicanuria, tuberculosis of lungs; blood-pressure 220.

*Case* 3.—Man, aged 45, had been in German army, but compelled to retire at 23 because of repeated vitreous hemorrhages in right eye; right eye was phthisical; left had cataract. After removal, retinitis proliferans was found but sufficient sight remained to enable patient to do moderate laboring work. Blood-pressure 185; casts, indicanuria, albumin, arteriosclerosis; patient reacts to tuberculin.

*Case* 4.—Man, aged 26; history of lost right eye at 23, retinitis proliferans. In left, vitreous hemorrhage. General condition: Blood, normal; blood-pressure, 110; hard radials, indicanuria; positive reaction to tuberculin, but no clinical tuberculosis. Under tuberculin treatment, and later potassium iodid, hemorrhage cleared and V.=20/30. Several months later it was learned through the mail that the left eye had gone completely blind. Cause unknown.

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[1] Arch. d'Opht., March, 1903; quoted in Ann. Ophth., July, 1903.

*Case* 5.—Man, aged 29, had first hemorrhage at 20 in right eye; several hemorrhages in a few months. Left eye attacked two years later. No hemorrhage for three years. Marked retinitis proliferans, but with sight good enough to enable patient to hold a semiclerical position. Blood-pressure was 115. Patient reacts to tuberculin; there was no clinical tuberculosis; Wassermann negative; marked and persistent indicanuria.

*Case* 6.—Man, aged 23, had first hemorrhage in left eye at 23; twelve since, becoming more frequent the past year. Tuberculin reaction positive; Wassermann negative; healed lesion at right apex; arteriosclerosis; blood-pressure taken by different men and with different instruments, constantly varied from 120 to 220; urine normal, but for very marked indicanuria. Patient was treated with small doses of tuberculin, two injections of animal serum, and later sent to the mountains, where he did well; gained in weight and had no more hemorrhages.

Bordley reports also having seen several patients with brain tumors, the extravasations absorbing; states that he has seen a great many cases but kept no notes.

Dr. H. B. Chandler, Boston: Four cases recently in private practice, all males, between 25 and 34. Three of them had one eye affected, one both eyes within two years. Nothing else wrong in eyes, and careful physical examination revealed no cause. Tuberculin tests not used, as from Chandler's experience in infirmary, all cases reacted positively. Blood-pressure taken in only one case; abnormally high. Treatment: Iodids, sweating and injection of blood-serum with improvement in vision in all cases.

Dr. Albert E. Bulson, Jr., Fort Wayne, Ind.: *Case* 1.—Man, aged 21, seen four years ago, was blind in one eye from extensive vitreous hemorrhage. Patient was under constant observation for year and a half. He had some fifteen or more hemorrhages during these years, one following the other so closely that for a long time fundal examination was impossible. Eventually the hemorrhages ceased, the vitreous cleared and left 20/15 V., which has persisted for two years. The most pains-taking clinical examinations were made without results, save pronounced indicanuria. After clearing of the vitreous, no lesion was found in the fundus.

*Case* 2.—A healthy young farmer had been under observation a year and had fifteen or twenty hemorrhages, each following rapidly on its predecessor. The fundus has never yet been seen. The man had some hemorrhages while in bed. He left the hospital and returned to his work. Recently he wrote that he had not had a hemorrhage for several weeks and he could read the headlines in the papers. The man has been subjected to rigid clinical and experimental tests, but nothing has been found save a persistent and excessive indicanuria. Both of these patients were treated with diaphoresis potassium iodid, etc. The reporter thinks the blood came from the ciliary bodies in both cases, largely because there was no evidence of fundal lesion, and the retinal vessels, when uncovered, were unaltered.

**Dr. John T. Carpenter**, Philadelphia: Patient seen in 1898 (only one in private practice), was a man, 30 years old, with intense neuroretinitis with retinal hemorrhages and exudates, veins swollen and tortuous, followed by recurrent vitreous hemorrhages, leaving vitreous dark. General health was excellent, but patient was an alcoholic. There was family history of gout. Patient was very constipated. Eye was totally lost, and reflex destroyed.

**Dr. E. A. Davis**, New York: *Case* 1.—Man, aged 30, was first seen for refraction error; V. 20/15 in each eye. A year later patient suddenly lost sight in right eye from vitreous hemorrhage on temporal side. Davis thought the blood came from chorioid, as at the upper end of the hemorrhage near the macula, the sclera showed through. Retina was partially detached at the temporal side and below. V. = fingers at 4 feet. In left eye there was subhyaloid hemorrhage just below macula, with a few fine retinal hemorrhages. V. = 20/20, in spite of lesions. Only systemic abnormality was traces of albumin and a few hyaline casts. Under treatment, nephritis cleared and hemorrhage was absorbed. In the right eye, which first showed extravasations, blood absorbed and retina reattached, with V. =20/30. Later retinitis proliferans with reduced V. = fingers. Left eye retained V. = 20/15 to last observation.

*Case* 2.—Patient was a woman, married, aged 22. Menstruation did not begin before 21, one year before being seen by Davis. Two days before menstruation V. in right eye was suddenly lost. It gradually returned until it reached 20/30. There was no lesion in the eye. When under observation the left vitreous showed a balloon-shaped hemorrhage V. = 4/200, becoming 20/200 after two months. Hemorrhage was thought to be of chorioidal origin.

**Dr. Allen Greenwood**, Boston: His patients have been between 20 or 40 years of age. None of them showed fundal or systemic abnormality. He describes the history of one patient as follows: "A watch-maker had over thirty hemorrhages, and frequently only light perception in one eye. The other eye had only one hemorrhage. The thirty-odd hemorrhages occurred during a space of eight years. Five years ago the patient was appointed to the police force, and left his sedentary life for an active one of constant walking. No hemorrhages since out-door work, and the eye of many hemorrhages now has a vision of 20/30, in spite of many vitreous strings."

DR. E. E. HOLT, Portland, Me.: Holt thinks that in most of his cases there has been nothing wrong in the eye save the hemorrhage, and that some form of nephritis was demonstrable in most of them. In a case seen sixteen years ago, with V. = 0.1, treated with "massage and cataphoresis," normal vision returned after a year, and was present fifteen years later. This was observed when the patient, a woman, came on account of a hemorrhage in the other eye, which recovered under the same treatment.

DR. B. R. KENNON, Norfolk, Va.: Kennon reports having had but one case, in a man whose first hemorrhage occurred at the age of 22. There were opacities from former hemorrhages when the case was under observation. Careful clinical search did not reveal constitutional lesion. Sight was gradually lost, each hemorrhage destroying a little of what was left.

DR. F. PARK LEWIS, Buffalo: Lewis has seen very few cases, and these before tuberculin tests, etc., were employed. A girl of 15 in perfect health, so far as clinical investigations indicated, had one single hemorrhage which resulted in retinal scars that damaged vision. She has since grown into robust womanhood. A boy had during adolescence repeated subhyaloid hemorrhages which destroyed the eye; general health remained unimpaired. He has grown to manhood without developing ill health.

DR. CHARLES H. MAY, New York: May reports three cases, patients aged 16, 20 and 36, all males. In all the eye structures were normal, barring the hemorrhages, and careful clinical study showed nothing. Two patients regained normal vision, one after a month, the other in a year. The history of the other is not known. Tests for tuberculosis were not made.

DR. H. MOULTON, Fort Smith, Ark.: Moulton reports a case in man of 60, with constant recurrences during five years, ending in blindness. General health robust at 70.

A second patient, 45, syphilitic, recovered normal vision in three months, and had no recurrence after six months.

Dr. Moulton adds that his youngest patient was 24, and most of them were between 40 and 60. Intra-ocular structures were normal, or, at the most, showed tortuous veins; often circumscribed patches of chorioretinitis. The majority of his patients showed positive clinical proof of syphilis. One case occurred in a cured lung tuberculosis, and no others showed evidence, though tests were not used. Arterial tension was, as a rule, high.

**Dr. Brown Pusey**, Chicago: Notes on two cases seen during past year. *Case* 1.—Patient was a man, aged 65; eye structures normal. Nothing is given about systemic examinations save blood-pressure, which was 200. Hemorrhage first in right eye, clearing in six months, with normal vision. Same experience four months later in left eye, and a second hemorrhage after same period in right eye. Patient recovered from all hemorrhages with normal vision.

*Case* 2.—Man, aged 43, seven years previously, had hemorrhage in left eye, which cleared, followed after six months by loss of eye from hemorrhagic glaucoma. First hemorrhage in right eye was one year, and second, six months before observation. Vitreous was full of opacities, and V. = 6/45. Blood-pressure was normal. Tuberculous pleurisy at age of 21. Family history of tuberculosis; office man, always constipated.

**Dr. Edgar S. Thompson**, New York: *Case* 1.—Man, aged 23; subhyaloid hemorrhage after pole-vaulting. Cleared up perfectly. Nothing in history or general clinical findings to explain hemorrhage.

*Case* 2.—Man, aged 55, had large subhyaloid hemorrhage in course of albuminuric retinitis. Eye was lost from glaucoma and enucleated.

Some of the men to whom I wrote gave their impressions from observation rather than detailed accounts of cases. Many of these are noteworthy. Bruns, of New Orleans, has seen cases in young adults with nothing else wrong. Some patients recovered, some lost the eyes.

Bull, of New York, thinks the subhyaloid variety usually develops destructive chorioretinitis; the vitreous, general hyalitis and uveitis. Patients of both classes are apt to show cardiovascular or renal disease, "if they live long enough."

Callan, of New York, says that in young patients the eye structures ars usually normal; in older persons there is arteriosclerosis. He thinks tests for tuberculosis unreliable unless other evidences are present.

De Schweinitz, of Philadelphia, excludes in his letter the hemorrhages in old persons from arteriosclerosis. Others occur between 18 and 30. Intra-ocular structures, he says, are often normal; nothing to account for hemorrhage. He twice discovered patches of chorioiditis. Regarding constitutional causes, he puts first, "so-called auto-intoxication as determined by the tests which I believe to be necessary and which are detailed in my paper on that subject before the Chicago meeting."[2] Tuberculosis, either clinically present or demonstrated by tests, seems to him an important causative factor, but treatment by tuberculin, in at least one case he sent me, was negative. Blood-pressure in these young patients was never high. Most cases went on to loss of the eyes from vitreous exudates, detached retina, secondary cataracts, vascular formation in vitreous, etc.

Ellis, of Los Angeles, says that cases in young people usually resulted in greater or less blindness from such causes as mentioned in de Schweinitz' note. General condition was usually good save for constipation, etc.

Friedenberg, of New York, has no data on tuberculosis. Syphilis was a frequent cause. Anemia with suppressed menses, dysmenorrhea, etc., have been associated conditions. Ocular structures were usually normal.

W. Reber, of Philadelphia, mentions four youthful patients, in three of whom the eye structures were normal. The other showed evidence of sclerosis in the retinal vessels. Three regained normal vision. The hemorrhage in the fourth case occurred while straining at stool.

Risley, of Philadelphia, has seen a few cases in children; thought the vitreous webs and chorioidal patches, which impaired vision, resulted from injury during parturition. Most other cases came in advanced life from cardiovascular diseases. Blood-pressure is usually high in this variety. Risley has never seen cases in which he thought it advisable to use tuberculin tests. Many young and clinically healthy adults recover.

Roy, of Atlanta, seems to have had experience confined to the sclerotic cases in advanced life.

Suker, of Chicago, seems to have had the same experience; his cases usually showed some permanent impairment of vision, attributable to the general condition.

Thorington, of Philadelphia, says that his patients were usually over 50. They improved only when the condition was due to syphilis. Arteriosclerotic or nephritic changes usually present.

J. E. Weeks, of New York, finds that cases usually occur between 15 and 22. Sometimes small venous hemorrhages in the retina precede subhyaloid or vitreous hemorrhages. Among causes he notes establishment of menstruation, constipation, irregularities of circulation. He has never used tuberculin tests. A girl of 16 lost both eyes from secondary glaucoma. In other young persons hemorrhages have recurred without serious impairment of vision.

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[2] Tr. Sec. on Ophth., A. M. A., 1908.

To the these cases, I add five which have come under my care recently.

*Case* 1.—Mrs. B., 29 years old, first consulted me in 1899 for asthenopia due to simple hyperopic astigmatism of 0.5 D. at 90°. She wore these glasses till she came to me again in November, 1909, to have them changed, as the eyes tired in reading. Vision, accommodation and muscular balance were normal. In both eyes were found numerous exudates beneath the retinal vessels; small extravasations were in each retina, obliterating the vessels, and careful examination showed small hemorrhages in the vitreous. She was sent to the hospital for clinical investigation. Dr. Gordon Wilson, clinical professor of medicine at the University of Maryland, was asked to make the examination, and I am indebted greatly to Dr. J. W. Robertson, now of Onancock, Va., and then hospital intern, for pains-taking observations. Only the following abnormalities were found: Decided accentuation of the second heart sound, and a transient albuminuria, with a few hyaline casts. The daily output of urine never exceeded 16 ounces. Blood-pressure was, on entrance, 240 mm. All the usual remedies were used without effect. Thyroid extract nauseated at once. Milk diet did the same. After a week I asked Dr. Arthur M. Shipley, of the surgical staff, to do a phlebotomy. He extracted 12 ounces of blood, with a blood-pressure reduction to 195 mm., the pressure returning to the old mark within twenty-four hours. Tuberculin tests, subcutaneous and von Pirquet's, were negative. There was not the least reason to suspect syphilis, and at that time I was not familiar with the Wassermann test. An interesting item in the patient's family history is that five relatives in preceding generations had died before the age of 40 from cerebral apoplexy. The patient went home; my diagnosis of nephritis was denied by her local physician, and we all came in for considerable criticism on account of our so-called "thoroughness;" this being the alleged cause of her equally alleged nervous prostration. The patient, however, lost her sight and a year later died.

This case does not, possibly, in the strictest sense, belong to my subject. Some of the small hemorrhages, however, were in the vitreous. Nor is it proper to draw a hard and fast line between various kinds of intra-ocular hemorrhage. The case illustrates one of the points made by Black in his report: the relation of high blood-pressure to the causative disease.

*Case* 2.—Mrs. T., aged 67, living in a small town in Maryland, was sent to me in December, 1910, for a change of glasses. Correction of low hypermetropia gave normal vision in each eye. Numerous small hemorrhages were found, apparently in the retina, near the right disk. The patient was not willing to stay in Baltimore long enough for a thorough examination, and I had to content myself with finding traces of indicanuria, not apparently excessive, and a blood-pressure of 220 mm. These findings were reported to her physician. She saw me again in February last. The old hemorrhages had absorbed, and there were many little ones in the vitreous, four to five diopters in front of the retina. The only thing in her history of note is a vesicovaginal fistula which was cured by operation. Outside of her right eye the patient seemed healthy; but I suspected that there was something she was unwilling to tell. I could not impress her with the possible gravity of the case, and she would not allow such tests as I wanted to use. At this writing, April 8, 1911, she is reported "well."

*Case* 3.—During December, 1910, a physician in country practice, 25 years of age, consulted me for sudden blindness in the right eye, coming on during a fit of coughing. A large subhyaloid hemorrhage covered the right foveal region. The retinal vessels could be traced to their usual visible limit, and were normal. No lesion, save the hemorrhage, was found. The left eye had 20/15 vision, and had nothing the matter with it. I had the patient examined by a competent internist, who gave him a clean bill of health with the exception of a blood-pressure of 210 mm., and "decided indicanuria." The man is an athlete, and looks in superb physical shape. He denied the possibility of syphilis or tuberculosis, and showed no indication of either. He was summoned to his home in the South before tests for tuberculosis could be used. A letter from him under date of March 3, states that the eye previously blind can now read large print, and is evidently clearing. I urged on him the necessity of running down the cause, if possible, but if he has undergone further examinations he did not disclose their result.

*Case* 4.—A boy, 15 years of age, worker in a wagon factory, came to my clinic at the Presbyterian Eye, Ear and Throat Hospital in October, 1910. He showed a left external squint, and stated that he had lost the sight of this eye some six months previously; that it had gone out suddenly, and he had never had the eye examined. The right eye was normal. Examination showed a cloudy vitreous, numerous large opacities and two or three vitreous hemorrhages, well anteriorly. Toward the nasal side could be seen a beginning retinitis proliferans. This boy was told to enter the hospital, but would not do so. I did not see him again until March. General examination showed nothing wrong save a possible slight splenic enlargement. There was some indicanuria, but no excess. Subcutaneous tuberculin test was followed by a violent local reaction and temperature of 100.5 F. There was no external local reaction in the eye, and I could not judge of the intra-ocular effect, if present. Following is the report of blood examination: Red cells, 4,700,000; leukocytes, 29,600; hemoglobin, 90 per cent.; polymorphs, 80 per cent.; small mononuclears, 15 per cent.; large mononuclears, 3 per cent.; eosinophils, 2 per cent. This is simply an inflammatory reaction. The retinitis proliferans shows increase. The general health seems unimpaired, and the patient is continuing his indoor work "until an outside job turns up." The family history gives no indication of syphilis; so, with positive tuberculous findings, a Wassermann test was deemed unnecessary. The fact that the boy was not in the hospital for a day or two before the tuberculin test must be considered in drawing conclusions regarding the febrile reaction. Blood-pressure, 120 mm.

*Case* 5.—Mrs. T., aged 29, came to me Nov. 15, 1910, for sudden loss of vision in the left eye. The immediate history was that on November 12 she had gone into the cellar to hunt up a reported leak, and had suddenly found herself in water up to her knees. Two hours or so later she knew that something had happened to her left eye. I found the vitreous absolutely black, save for small areas, down and up, which gave a fair reflex. The patient was subjected to a careful physical examination by Dr. Wilson. She was the mother of two children and had had one miscarriage—her first pregnancy. She weighed 140 pounds and looked in excellent health. Menstruation had always been regular, and she had nursed all her children. Syphilis, tuberculosis and intestinal toxemia were excluded by examinations and tests. She was not menstruating when the eye hemorrhage occurred. For want of better indications in the way of treatment, she was put to bed in the hospital, given pilocarpin sweats and potassium internally. After two weeks the vitreous hemorrhage had cleared sufficiently to enable me to see a large subhyaloid hemorrhage over the fovea, and extending nasally over the disk border. A few smaller subhyaloid hemorrhages were seen at the nasal and lower edge of the disk. All of these gradually cleared, the foveal extravasation being the most persistent, while the disk margins were entirely free after a month. On January 19, the patient had central vision of 8/200, and could trace the perimetric disk in all parts of the field. On February 9 I observed a recurrent hemorrhage over the upper and outer disk margin. Blood-pressure varied between 115 and 130 mm. After the second hemorrhage in February, I urged the patient to have some teeth cared for, to which Dr. Wilson had called my attention as a possible portal of entry for infection. What influence, if any, this had I cannot say; but late in March central vision had increased to 20/100, indistinctly, and there had been no further hem-

**orrhages. As study of the fundus became possible, the uncovered retinal vessels came out normal.**

### DEDUCTIONS

1. *Results.*—So far as the above collective observations admit of analysis, from the standpoint of results, the following is approximately true: Of forty-eight eyes, twenty recovered with practically normal vision to the time of last observation. Seven obtained more or less improvement, the nature of the residual lesion not being given. Seven developed retinitis proliferans, three of these being totally blind, the others preserving sight enough to do some work. The other fourteen were totally lost, four from secondary glaucoma, one from phthisis bulbi, and nine from causes not specified. probably vitreous opacities, retinal detachment, etc. Seventeen out of the forty-eight eyes were lost.

2. *Recoveries.*—A number of cases in the foregoing reports and in literature, showed one hemorrhage, or recurrent hemorrhages, producing temporary and complete blindness. Then normal vision was slowly restored and retained. Nothing was found in the eye to account for it. The source of blood is generally ascribed to the chorioidal vessels. This is based on the facts that the retinal vessels, when they can be seen, as in the subhyaloid variety, show nothing wrong, and after absorption of the blood, the same is true. It is hard to think of normal retinal vessels being responsible for so much hemorrhage. A still stronger reason is found in our knowledge derived from recent studies in chorioidal diseases. We know that many pathologic conditions produce exudative chorioiditis. Intestinal parasites, "indigestion," chronic appendicitis, and malaria, may be mentioned. We attribute these ocular lesions to toxins or organisms in the blood, and their manifestation in the chorioid to the large vascular area therein. It is not going very far to imagine the same influences breaking down a vessel and producing hemorrhage. So long as vascular resistance is less than the acting cause, recurrence persists. When the cause is removed, through either intelligent diagnosis and treatment or general hygienic management, hemorrhages cease. Note, for instance, the cases reported by Greenwood and Bordley. Recovery of vision must depend on the harm done by

. and recurrence must depend on
ntal removal of the cause.

*ystemic Conditions.*—Blood-pres-
ark is apparently borne out by the
n high, it is usually associated with
riosclerosis. In each we have conditions
ated to produce hemorrhage through a
tis or phlebitis. Bull believes that such
s develop arteriosclerosis or nephritis, if
ng enough. One of my own patients was
ar of nephritis; yet no one would have made
gnosis on this young woman on the urinalysis
oms alone—outside of the intra-ocular hemor-
nd blood-pressure. C. S. G. Nagel[3] reports a
which retinal hemorrhages were the first symp-
diabetes. (I call attention here to the previously
ssed opinion that we must not differentiate intra-
ar hemorrhages too closely according to their situa-
n.) The patient was apparently in good health.
gel notes Leber's observation that retinal lesions occur
ly after years, and when emaciation and general
decline are present. Webster Fox[4] discusses the relation of high pressure to intra-ocular hemorrhages, and shows the close relation between them and various forms of nephritis. In the article by Scrini and Bourdeaux,[1] the statement is made that these essential vitreous hemorrhages have no prognostic effect on general health. I think that this statement is to be accepted with allowance. Nagel's diabetic, and my first case are illustrations. My third belongs to the class which must be regarded with concern. Something caused the high arterial tension, and the fact that we do not know what it was in no way proves that it is not serious. I cannot extend the paper to quote other authors, but I am convinced that these hemorrhages, accompanied by high pressure, ought to be regarded as a possible early indication of chronic nephritis. But they occur, too, with low pressure. Black expresses the opinion that dilated heart or auto-infection is at the bottom of these. We still have the dilated heart to account for. Usually some systemic infection precedes this. Of course, the passive venous congestion, might be a mechanical cause. As to auto-infection, i. e., absorption of either the organisms

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[3] **Nagel:** Ophth. Rec., October, 1904; abstr. in Ann. Ophth., January, 1905.

[4] **Fox:** Ophth. Rec., October, 1908.

themselves or toxins, which might act deleteriously on small or terminal vessels, I believe that here we are approaching the main etiologic factor in most cases, and that careful search for the nature of the infection may often lead to the diagnosis of a condition giving no other symptom.

Axenfeld and Stock[5] report a case of a woman, 30 years of age, with a vitreous hemorrhage of the right eye. The left eye was normal; but several weeks later a grayish-yellow exudate appeared in the periphery of this eye. There was also a perivascular exudate along a retinal artery which ran toward the chorioidal focus. There was both general and local reaction to tuberculin in both eyes. "During the treatment with small doses of tuberculin the foci in the chorioid and perivascular exudate completely disappeared, and in the right eye, in which in the meantime an indolent posterior synechia appeared, the vitreous cleared. One must accept that in this case a chronic tuberculous uveitis had begun with the appearance of a simple hemorrhage in the vitreous, which surely had arisen from the retinal vessels." It seldom happens that one can observe such a sequel of events in these cases.

De Schweinitz and Holloway[6] report a remarkable case of retinal hemorrhages in a boy of 20, who gave a positive reaction for tuberculosis. The late Dr. Kipp, of Newark, reported an instructive case[7] in this connection. I call attention to my fourth case, which showed positive tuberculous reaction without symptoms outside the eye. Such observation seems to me to show that these cases, even if the eye gets well, must always be viewed with suspicion, and that it is best to treat them as though the diagnosis was made. Greenwood's policeman (given in this paper), and Bordley's sixth case are examples.

Indicanuria plays a large rôle in these concomitant systemic conditions. There is, I know, discussion regarding its value as a diagnostic sign; but its persistent presence in excess is generally thought indicative of disturbance of metabolism. It was observed in most of Bordley's and in Bulson's cases. It was seen in one of mine, but not in sufficient quantity to make us think it significant. The frequency of obstinate constipation in the description of these cases leads one to think that indican would be found more frequently if always looked for. It occurs without reference to blood-pressure. Where the pressure was high, as in Bordley's cases, there were other conditions, usually renal, to account for it. The close association of indicanuria with functional and organic uveal lesions has been presented to this Section too recently to need repetition. Vitreous hemorrhage, with tuberculosis (sometimes clinically undemonstrable) shows that this form of infection can produce vascular diseases in the eye. Indican in the urine means intestinal putrefaction, and this means not necessarily absorption of putrefactive toxins, but malassimilation, allowing absorption of other toxins, which, under better conditions, the system might avoid. Here we come against other concomitant conditions. Anemia, unless essential, must have a cause. Dysmenorrhea, amenorrhea, unless due to local conditions, often admit of an explanation which involves nutrition. They are but symptomatic of the same disturbance which causes the intra-ocular hemorrhage, and are not themselves a cause.

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[5] Axenfeld and Stock: Klin. Monatsbl. f. Augenh., January 1911, p. 28.

[6] Univ. Penn. Bull., March, 1910.

[7] Arch. f. Ophth., v, 38.

To discuss treatment of these hemorrhages is impossible, save to say that for the most part oculists have been using diaphoresis, the iodids, etc. It seems to me that we should view these cases about like this:

1. In older people, i. e., persons over 50, we usually have signs of arteriosclerosis, and if they are not present, the hemorrhage itself is evidence. Eliminating special causes of vascular changes—syphilis, nephritis, etc.—we have to manage, as best we can—or the internist has to manage—the vascular changes. Along this line, we will get our best results.

2. In young people, whether there is a single hemorrhage or many, we are dealing with some infection or disease capable of producing vascular changes. High blood-pressure throws suspicion on the kidneys, whether or not there are other symptoms of nephritis. Anemia, menstrual disturbances, etc., are but symptomatic of malnutrition probably due to an infection. This may be tuberculous, and may be of some other kind, brought about through defective metabolism. Its presence and nature is to be determined by rigid laboratory examinations.

3. Even when we can find no cause, and have to resort to the "alterative" treatment, we should bear in mind

that the cause has not been determined; that it is usually of a serious nature, and that it is well to give the patient the benefit of doubt with regard to the three causes apparently most apt to be found—tuberculosis, defective metabolism, nephritis.

842 Park Avenue.

## MEMORANDA

# REPORT OF THE COMMITTEE ON COLLECTIVE INVESTIGATION CONCERNING THE OCULAR MUSCLES *

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HOWARD F. HANSELL, M.D.
AND
THEODORE B. SCHNEIDEMANN, M.D.
PHILADELPHIA

In view of the varied character of the observations reported last year, and in view of the studies which have been commenced since then, some of which are completed, it becomes necessary to use most of the space allotted to the report this year in arranging the data which we have, and in making clear the general plan of this investigation.

Thus in the spring of 1911 this Committee finds that it has (1) the data relating to apparently disconnected points of anatomy and physiology which were summarized in the last report; (2) communications of the same kind which were either necessarily left out of that report or have been submitted more recently; and (3) other data which have also been obtained since, and which relate only to the physiology of the muscles or to anomalies of accommodation.

Now it is evident that a collection of isolated facts, however new or important, is of little use unless the relation of the facts to each other is made apparent. If this investigation is to be of interest to those who take part in it, or of any value later, there should be a definite plan of procedure. At least, some arrangement of the data should be made with relation to each other. Therefore, the Committee deems it advisable at this point to state clearly the plan which seems most direct, and the relation of these facts to each other, even though the attempt to make this statement may monopolize a large part of the space which can be allotted to a report in any one year. With such a clear understanding now,

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

it will be possible to advance later much more intelligently and rapidly.

It may be said, in a word, that while our endeavor would naturally be to clear up first any obscure points in anatomy and physiology of the muscles, we must also have a certain plan in doing even that part of the work. And as the relation between accommodation and convergence is so intimate, a knowledge of the intra-ocular muscles must form the foundation of a study of the extra-ocular groups. Having done this, the next step, obviously, would be to study the motion of one eye alone (the non-associated movements), both eyes at rest, and finally both eyes in motion (the associated movements) either without or with accommodation, with convergence, or with torsion, or with all three together. Having cleared up all the doubtful points in these various aspects of the subject as they pertain to the normal eyes, we would then be better prepared to approach simpler problems of the pathology.

This might be called the direct line of the investigation, and until some better plan can be suggested it seems the best one to keep in mind. It will serve at least as a line on which the desired data can be hung as they may be cleared up one after another.

Now this Committee still believes that the plan of study thus outlined is the one which will ultimately give the most satisfactory results, and would strongly advise persistence in it; but the experience of the past two years has shown that it is useless to expect any considerable number of men to undertake real investigations which require special reading, considerable patience, and usually much time. Many members of the Section, forgetting the unsettled condition of several underlying questions of anatomy and physiology, are ready to start at once with what they call "a practical question" of treatment. Again, a number of the members are quite ready to confine their attention for a few years to certain aspects of the anatomy or physiology of the muscles and have done so with true scientific enthusiasm, but they had no special interest in making the tiresome measurements which are necessary in any study of normal or abnormal accommodation. Indeed, the number who persist in that are few.

In other words, we find that we have before us not only the line of investigation which has just been outlined, and which has been called the direct line of study, but also what may be called an indirect line. The latter are none the less valuable data in the right place. But they are data more or less remote from those topics which we would naturally study at first. It is for this reason that the results of the last year may at a first glance appear detached from each other, demanding an arrangement now.

In the future also other data will undoubtedly be contributed which at a first glance seem quite detached from any others. While some members of the Section are still studying questions of physiology or even the anatomy of the muscles, others may have begun with questions in pathology. If these results sent to our Central Committee are really facts, and new facts filling up the gaps in our knowledge, such reports will be gladly accepted. When, however, the Central Committee makes its annual report to the Section it will endeavor to follow first the direct line which has here been indicated as the most natural for us to pursue, and after that, and quite independently, will report any other new facts relating to the muscles which belong not to the direct but rather to the indirect line of the study.

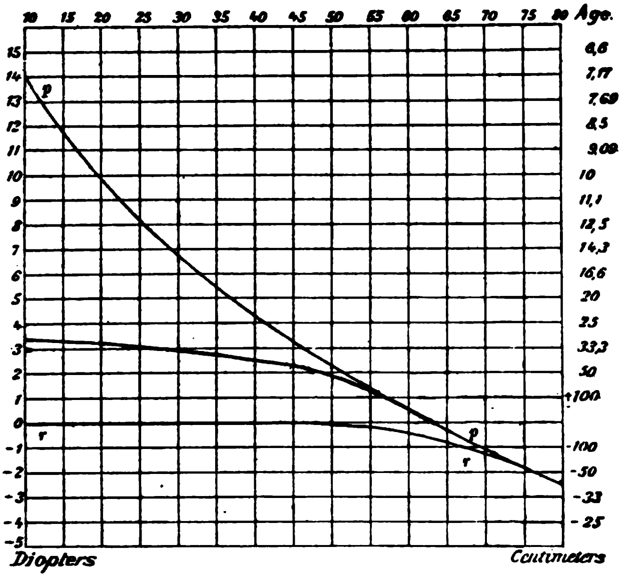

Fig. 1.—Representation of the change in the position of the far and of the near point with advancing age as given by Hess, together with the curve found by others to represent the decrease in the amount of relative accommodation with parallel axes, also with advancing age.

With this explanation of the plan which has been or which will be pursued, we are prepared to note a few points made prominent by our studies during the past year or previously.

### NORMAL RANGE OF ACCOMMODATION AT DIFFERENT AGES

Commencing with the muscles of accommodation we can fortunately pass over most questions of anatomy, since these are already quite well established. Nor is it important for us to determine exactly what changes take place in the lens itself during the act of accommodation, that still being a question of difference between such careful students as Hess and Tscherning. Fortunately, it is sufficient for practical purposes for us to agree that accommodation is essentially an active effort of the ciliary muscle, no matter what shape the anterior surface of the lens may assume or how that is done.

It *is* very important for us, however, to agree on the curve which represents in normal eyes a decrease in the range of accommodation with increasing years, and also to know the decrease in the positive part of the relative accommodation with parallel visual axes (that is, of course, the decrease in the ability with parallel visual axes to overcome minus lenses) in proportion as we grow older.

In regard to the curve which represents the accommodation of the normal eye at different ages, it is true that the original curves given by Donders have been modified somewhat by the observations of Hess, by Duane, and others. But the differences are slight, depending somewhat on the method used for making the measurements. As the curves given by Hess in the last edition of Graefe-Saemisch have been accepted by the very large majority, the Committee would recommend that these be considered as representing the range of accommodation in the normal eye with increasing age. These curves are reproduced as part of Figure 1. In that figure also there is included the curve which represents the decrease in the positive part of the relative accommodation with increasing age.

### INFLUENCE OF DRUGS ON THE ACCOMMODATION

Having agreed on these well-known curves as those which represent the changes which occur in the range of accommodation of the normal eye, the next question is to ascertain what effect is produced on the accommodation when definite quantities of certain drugs (for example, small amounts of homatropin, physostigmin, etc.) are applied to the conjunctiva of individuals who have normal eyes, but who are of different ages.

This is an aspect of our subject to which but little attention had been paid, but which is undoubtedly of importance for three reasons. These are:

1. The promptness and the degree in which a cycloplegic or miotic acts on normal eyes is apparently in proportion to the amount of the dose, up to what may be called the normal effect of that drug.
2. This action of the drug assists us in determining whether, in a given case, the muscle of accommodation seems to relax or contract (a) in a normal manner; or (b) whether that relaxation or contraction is more rapid than usual, indicating thus a tendency to insufficient accommodation; or to excessive accommodation.

3. A third and very decided value of these measurements of the effects of the cycloplegics and miotics is that as these drugs are used more and more clinically, it becomes evidently desirable to know what their effect is on normal eyes.

In the report last year attention was called to the work which had been done by Lewin and Guillary in their classic "Die Wirkungen von Arzneimitteln und Giften auf das Auge." But there still remains a number of observations to be made, in order to fill properly this gap in our knowledge. Last year, also, a summary was given of the contributions in this direction by Drs. Marlow, Sulzer, Martin and Crockett, all members of our Section.

### OTHER STUDIES OF CYCLOPLEGICS

As for the studies in this line reported to the Committee since 1910 we should call attention to the following:

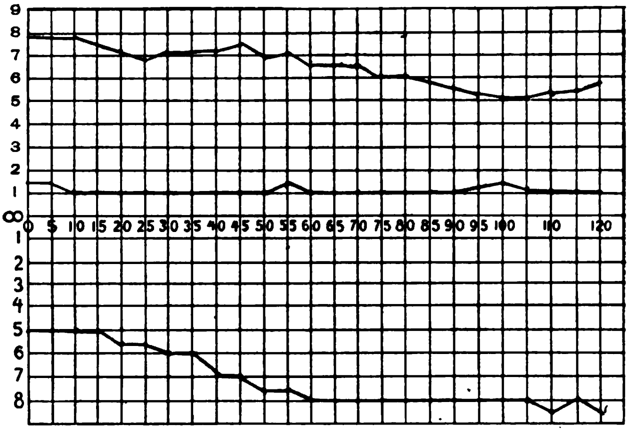

Fig. 2.—Effect on a normal eye of the least dose of atropin sulphate (about 0.0000013 gm.), which will produce dilatation of the pupil (Drs. Charles A. Oliver and Elmer E. Johnson of Philadelphia). In this diagram the upper line represents the effect expressed in diopters on the near point. The next line represents the effect on the far point. The lower line represents the diameter of the pupil expressed in millimeters.

1. Measurements of the effect of small doses of atropine sulphate by Dr. Elmer E. Johnson, under the direction of Dr. Charles A. Oliver of Philadelphia.

In these experiments Dr. Johnson did not use disks, but Dr. Oliver mentions that special care was taken to equalize the size of the "drops," and this, with the evident care in the measurements, probably accounts for the rather constant results.

Sixteen such experiments were made on as many different individuals. Measurements of the size of the pupil and of the range of accommodation were taken on the average about every two minutes. In other words, we have a total of 968 different observations.

In a report of this kind it is possible, of course, only to summarize very briefly the results of work which has often demanded much time and patience. Thus it is practicable in this connection only to reproduce a few illustrative curves and refer to some of the salient points which the measurements have brought out. These are:

1. There is a certain minimum amount of the drug which does produce dilatation of the pupil, but which does not produce any very marked effect on the accommodation (Figs. 2 and 3).
2. The dose for this, found by Drs. Oliver and Johnson as the minimum amount of atropin which produces relaxation of the accommodation, is in general about the same as that which has been found by others to produce that effect. The importance of this observation is much greater than would at first appear, if only as corroborative evidence. For by placing this practically minimum dose of atropin in the conjunctival sac of an eye, then measuring the rapidity and the degree of the relaxation of the accommodation, we can judge, at least approximately, whether we have to do in that eye (a) with a normal accommodation or (b) with one in which there is a tendency toward insufficient accommodation, if the relaxation is too prompt and too great, or (c) with a tendency toward excessive accommodation, if that relaxation is tardy or slight.

## STUDIES OF ANOMALIES OF ACCOMMODATION

As the fundamental principles of normal accommodation have now been quite well established, and as anomalies of accommodation were those which would come next in order of investigation, it was suggested that at least a preliminary study be made of these variations from the normal. At first glance that seemed simple enough. But when our members found that it meant patient measurements frequently repeated, the greater part gave up the plan for the present, though expressing the hope that the work could be carried out in the future. Therefore, it does not seem warrantable to give more than this word of even a preliminary explanation. But the plan of study which was outlined on page 111 of our last report still seems the one best adapted for our guidance. Moreover, it may be added that a sufficient number of measurements have been made on the eyes of suitable subjects to show the importance of anomalies of accommodation, and it can be asserted with confidence that another year will enable us to report data concerning this aspect of the question which are apparently of decided clinical value.

### OTHER OBSERVATIONS IN THE DIRECT LINE

Other observations in the direct line of this inquiry have been submitted during the past year, and some are almost ready for presentation to the Section. But as justice could not be done to them in the limited space which remains in this report it is better to refer now even briefly to observations not in the direct line.

### OBSERVATIONS NOT IN THE DIRECT LINE OF STUDY

These relate to questions pertaining more or less directly to the physiology of the extra-ocular muscles. Foremost among them, acknowledgment should be made of a communication from Dr. G. C. Savage of Nashville, Tenn.

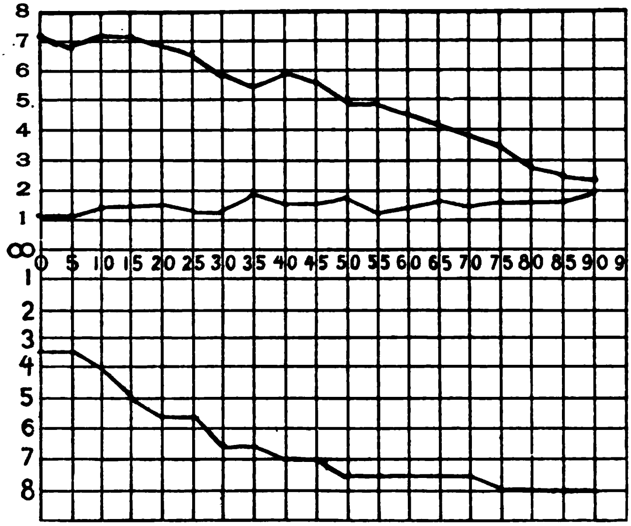

Fig. 3.—Effect on a normal eye of the least dose of atropin sulphate (about 0.00001 gm.), which will produce relaxation of the accommodation (Drs. Charles A. Oliver and Elmer E. Johnson of Philadelphia). In this diagram the upper line represents the effect expressed in diopters on the near point. The next represents the effect on the far point. The lower represents the diameter of the pupil expressed in millimeters. The second curve indicates that some change might have taken place in the position of the far point. This must be due to the indefinite replies of the subject, for the reason that atropin never causes the far point to approach. But the curves are plotted from the figures just as they were given.

### THE POSITION OF THE POSTERIOR POLE

When this committee was preparing the questions for study, one of those chosen related to the position of the posterior pole. The principal reason for this selection was that Dr. Savage was much interested in the subject, had written concerning it, and as a matter of courtesy, and with the hope of obtaining new data, he was asked to be the chairman of a subcommittee, associating others with himself.

Considerable correspondence followed, he suggested no associates, but on Feb. 17, 1910, he sent to the committee his "final report" in the form of a reprint of an article on that subject which had been published in the *Ophthalmic Record*, in December, 1909.

Last year no report on this part of the subject was made by the committee, for, although it might be quite in order to mention original communications already printed, on the other hand, to attempt even a brief digest of such publications would necessarily increase the length of the report, and doubtless give offence to some who might consider their articles slighted. Such abstracts of articles already printed can be found easily in various journals.

Moreover, our report last year had already been prepared, and was quite a long one before this reprint was received.

As Dr. Savage, however, is one of the oldest members of our Section, and as he has devoted much time to this question, it is proper that this committee should do more than simply acknowledge the receipt of the reprint. Therefore, we wish to state that the gist of the contention is given in the words of the author as follows:

"In Figure 3 I have shown Helmholtz's so-called optic axis o-a, and on it his nodal point, useless each e'en with the others. The sooner they are forgotten the better, and the angle gamma should be included in the forgetting. The true posterior pole is the central point of the macula, the true optic axis is the visual axis, and the true anterior pole is that point of the cornea cut by the visual axis, whether it be the center of the cornea or not."

As it is the object of the committee to avoid the discussion of theoretical questions, and to confine the report as much as possible to facts—preferably facts which can be expressed in numbers or by photographs—it is not deemed wise to argue the point at issue. We can only refer members of the Section to the article, and to the other writings of Dr. Savage without any expression of opinion, except to say that the demonstrations made by Helmholtz, and by the long list of students of physiological optics, and which have been up to this time so generally accepted, still seem to the committee to be those which its members also are inclined to accept.

## TORSION

Acknowledgment should also be made of observations of the torsion which occurs in normal eyes in the primary position. These measurements were made by Dr. Frederick Frisch on the eyes of soldiers at Fort Porter, with the clinoscope of Dr. Stevens. Although the data are interesting, they still require further verification, and that, with the length of this report already, makes it necessary to defer the presentation of that subject this year.

### CONCLUSION

Considering the short time which has elapsed since these studies were undertaken, the results seem quite as creditable as could be expected.

The vitality of the Section cannot be shown in any way better than by the readiness of its members to cooperate in special lines of study. National ophthalmologic societies of other countries have long ago set the example of appointing certain committees for the collective investigation of certain subjects. It is generally admitted by our foreign colleagues that more attention is given to the study of the ocular muscles in America than any where else. Therefore, it is hoped that by patient and combined effort this Section of our national Association may ultimately clear up some of the difficulties of this obscure subject, for our own credit and for the benefit of others.

### RECOMMENDATION

In view of the foregoing, the committee recommends:

1. The studies already undertaken should be continued along the lines indicated in this report.
2. Cooperation should be invited again of all members of the Section who may care to take part in the investigation.

# MEMORANDA

# THE OPTOMETRY QUESTION AND THE LARGER ISSUE BEHIND IT *

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DENVER

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Before they were appreciated as an agency for the prevention and cure of disease, glasses had been used for five hundred years. The optical properties of lenses and of the eye had been worked out by physicists, Galileo, Keppler, Gauss, Brewster and others. Skilled opticians had met every demand made on them to correct the optical deficiencies of the organ of vision. Yet only a small minority of elderly people put back the limitations of age by wearing spectacles for reading. Perhaps one myope in fifty had extended his range of vision by concave lenses. The royal astronomer at Cambridge, England, and a philosophic clergyman in New York, with the help of a Philadelphia optician, had worked out corrections for their own astigmia.

Meanwhile Milton, Eulenberg and many other scholars went blind from eye-strain. Pepys, at the age of 37, laid aside his inimitable diary, giving up a hopeless struggle with hyperopia, and preparing for blindness which never came, though he lived thirty years afterward. Educated Germany grew largely myopic, and the great mass of suffering from eye-strain and disability caused by ametropia went wholly unrelieved.

But sixty years ago a trained physiologist and physician, Donders, became interested in the optical defects of the eye. Other men trained in medicine took up the subject—Snellen, Knapp, Dyer, Weir Mitchell and their successors. The influence of errors of refraction and their correction was seen from a new point of view, and the use of spectacles was enormously extended. To-day

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more lenses are worn for correction of eye defects in a single city than were used sixty years ago in the whole world.

From the imperfections of the human eye that disqualify it for meeting the requirements of modern life, and the ability to give relief by properly chosen lenses, there has developed a great industry engaged in supplying the mechanical aids to vision, and a great opportunity for professional service in bringing to each individual the peculiar assistance required. As in the opening up of any new field of activity, men have rushed into this one to exploit the opportunities here offered. Among the medical profession a certain number have appreciated this field for service, and the rewards for such service. On the other hand, among those who approached it from the non-medical, the mechanical and commercial sides, many have perceived its richness. Each party has pushed forward, proclaiming its superior right, by priority and fitness, to the field in question, like prospectors in a new mining camp. In this way has arisen the optometry question, which now seems to be reaching its most acute stage.

In discussing this question two important facts should be kept in view.

1. The process of estimating the refractive condition of the eye is a distinct technical process. It is as different from a chemical analysis, a microscopic examination, a physical exploration of the chest, a skilful combination of drugs, or a capital operation on the brain or abdomen, as these are different from one another. Ability to measure the refraction of the eye is no more to be presupposed from ability to do these other things than is ability to remove a brain tumor to be presupposed from a possession of skill in chemical analysis.

2. Errors of refraction and their measurement are of general interest because of their broad general influence on health. Until this influence was recognized the economic importance of spectacles, or of their adaptation to the eye, was insignificant. All attempts to escape this relation of glass-fitting to health, and to the departures from it that constitute disease, will fail. Bearing in mind these two facts, the special character of the process of measuring the refraction of the eye, which may well entitle it to a distinctive name—optometry, and the clear inclusion of optometry within the circle of

the healing arts, let us look at the present situation with regard to it.

### THE OPTOMETRY LIE

In twenty-six states of this country and three provinces of Canada there are so-called "optometry laws." All of them are based on the supposition that the practice of optometry is actually and properly something apart from the practice of medicine. They assume that a person may be qualified to practice optometry who possesses but a superficial and theoretic knowledge of the eye, and who is ignorant of the structure, workings, and diseases of all other parts of the body. Let us discuss the subject on the basis of this theory, ignoring for the time the practice which, under each of these laws, has conferred the advertising advantages of a state license on all who have been engaged for a certain period in the optical business, without even such limited knowledge as these laws assume to be necessary.

The most able and brilliant champion of this theory of optometry, recognized as such by optical associations and journals, Mr. C. F. Prentice, president of the State Optometry Board of New York, has recently put forth a challenge to the editors of the medical journals. It includes seventeen statements, which he calls "tersely stated truths." These so nearly express the thoughts and claims of optometrists in general that some of the maxims have been adopted as mottos to be displayed on buttons at their optical meetings, in their advertising, and in the campaigns before the legislatures.

The first of these statements is "Physics is not physic." This is one of those epigrams, containing the literal, insignificant truth, which best serves to hide the larger falsehood implied in the use made of it. The words "physics" and "physic" are derivatives of the same Greek root, with history and meaning in common. They have become differentiated, the one to mean the science or sciences of the phenomena associated with matter in general, the other to mean the application of the resources of the material world to the relief of disease and suffering. Physics, like chemistry, may be so applied in the service of "physic" or medicine; indeed, to refuse the aid of physics to medicine would be as absurd as to deny medicine the aid of chemistry. The shaping of a lens, according to the laws of optics, belongs no more peculiarly to physics than does the shaping of a brace or splint; and either may be as true and legitimate a curative agent as a serum or a drug.

The second of these "tersely stated truths" is, "A lens is not a pill." No, it is not a pill; neither is a pill a hypodermic syringe, or a clinical thermometer, or a stethoscope, or a collyrium, or an operating-knife. But a lens, like all of these, may be used to prevent or cure disease or relieve suffering. Neither the lens nor the pill are of general interest simply because they are solids of a certain limited size, specific gravity, or color. They are of general interest because they may be made to prevent or cure disease or relieve suffering; because they are efficient means of accomplishing what men in general desire to accomplish. And for this reason they have equal claims to recognition in the armamentarium of those who practice the healing art.

Then Mr. Prentice goes on, (3) "A lens treats light"; (4) "A lens does not treat disease." The lens itself exerts the same influence on light in the engineer's transit, the astronomer's telescope, the microscope, the opera glass, or the photographic camera. In each of these uses of the lens some men are interested. But the general important interest that has thrust the optometry question into the arena of heated discussion and legislative action is the application of the lens to the relief of suffering or the treatment and prevention of disease. So when Mr. Prentice goes on to say (5) that "Optometry is not medical science," and (6) that "Optometry is founded on optical science," he has put himself pretty fairly in opposition to the facts he is trying to deal with.

Using the term "medical" in the broader sense in which it is generally used, and which every intelligent man understands, he would be far nearer the truth if he were to say that optometry is only a part of medical science. Optometry is the application of optical science to the purposes of medical science. But enough has been said to show the basis of independence and separation from medicine on which optometrists have claimed, and to some extent have secured, recognition as a distinct pseudo-profession.

To illustrate the falsehood of that claim one does not need to go beyond their own utterances. In the same number of the *Optical Journal and Review* from which Mr. Prentice's challenge has been quoted, I find in the first article what purports to be a statement of the permanent action of atropin on the accommodative function, "which our friends the oculists have not noticed." There is a paragraph as to diagnosing disease by the iris, which takes seriously a scheme of quackery that has arisen in Germany. For changes in the refraction of a case it is editorially suggested, "Possibly there is some pathogenic condition within the retinal blood-vessels which has caused them to swell at times." And for eyes that see print of different sizes, "It looks very much like a pathologic condition of the eye whereby the rods and cones are being forced apart, perhaps by some growth behind them." The address of Mr. Parsons before this Section last year is quoted *in extenso.* Of course, the medical opinions in any way adverse to the use of cycloplegics are paraded as completely settling the practical question of the use or non-use of those drugs. One of the articles is headed, "The Cooperation of the Patient"; and another, "How to Please your Patient"—not "customers" or "clients," but "patients," the term always used as correlative of physician or surgeon. One correspondent urges that the optometrists "should be allowed to use all means in order to make a proper diagnosis." "With a two or three years' course in a reputable college, optometrists will easily master the pharmacal, clinical, and therapeutic value of the very few drugs necessary in eye work." Another correspondent discusses the physiologic action of atropin, making comparisons with that of alcohol. It is not surprising also to find here that the Rhode Island Society of Optometry has decided, "that optometrists are justified in using the title of doctor, that it is a benefit to them from a commercial standpoint, and that it tends to elevate the profession."

The favorite definition of optometry in the laws relating to it is: "the employment of any means other than the use of drugs, for the measurement of the powers of vision and the adaptation of lenses for the aid thereof." Some of the laws require examinations in the anatomy and physiology of the eye. Others leave the scope of the examination entirely to the Board of Examiners. The discussions in the societies of optometrists and their advertisements show keen appreciation of the medical bearings of their work.

In their actions, which speak far louder than the words in which they plead for special legislation, the optometrists show that they understand that optometry, as they wish to develop it and practice it, is a part of the general practice of medicine. In their argument for the legal recognition they desire, they quote as a pertinent conclusive predecent, the profession of dentistry and its legal status; and when they do this they raise the true question, the greater issue behind optometry.

## SHALL THERE BE ONE OR SEVERAL PROFESSIONS OF MEDICINE?

This is the question that is facing, not only the ophthalmologist, but equally the whole medical profession and the public. No true profession will ever be permanently established on the narrow basis on which optometry legislation has been advocated. The measurement and correction of errors of refraction for the relief of symptoms will be associated with a knowledge of symptoms, and of the causes of symptoms, as well as of the general knowledge of the structure and functions, of the organs in which these symptoms arise, and of the application of other means to their relief.

In deciding whether it would be better that the body of workers engaged in the practice of different branches of the healing art should be divided among separate professional bodies, or if it would be better to have them in the specialized groups of a single profession, with common educational requirements, professional associations and ideals, the precedent of dentistry will have an important influence. It is generally admitted by the leaders of professional thought among the dentists, and by those who have given attention to the organization of the medical profession, that the separation between medicine and dentistry is to-day too wide for the best interests of either profession, or of the general public. Physicians should know more of dental pathology; dentists should know more of medicine. Dental organizations are endeavoring to raise the educational requirements for their profession more nearly to those of the medical profession. The recognition of dentistry as a specialty of medicine in our national medical organizations, especially in the Section on Stomatology of the American Medical Association, seems to indicate that the need and advisability of a profession of dentistry, as a separate profession apart from medicine, is by no means established.

It cannot be denied that dentistry as a separate profession has shown a remarkable development, and has accomplished great results in the prevention and cure of disease and the relief of suffering. It is better to have dentistry as a separate profession than not to have it at all. And to achieve its present development in the period in which it has developed, it may have been necessary that it should free itself from the trammels that might have been placed upon it by the leaders in the medical profession, who were not sufficiently informed or interested with regard to its needs to render it any valuable assistance.

But in this respect the case of ophthalmology is quite a different one. The development of ophthalmology has been somewhat hindered by the professor who insisted that he could teach all it was necessary to know of diseases of the eye in two of his surgical lectures; and by the men who, as students, listened to such inadequate presentation of the subject, and who now as leaders and seniors in the medical profession fail to appreciate either the extent or the practical value of ophthalmic science. But even in the particular branch of optometry development has been wholly within the medical profession, from Helmholtz and Donders to George T. Stevens and George M. Gould, the advances in optometry, and even the popularizing of it have been the work of men within the medical profession.

The optical journals to-day mix their articles on lens-grinding, frame-fitting, window-dressing, and newspaper advertising with quotations of all articles written by members of the medical profession bearing on optometry. The medical journals are still the chief source of this part of their literature, which is supplemented by articles written by men of medical education who find their chief sources for practice and revenue among the optometrists. Only since they started out to convince the legislatures that optometry was physics and not physic, have these journals begun to call on professors of physics for statements of those laws and facts of optics which have an established place in the annals of mathematical science alongside the propositions of Euclid. The books which are advertised to the optometrist and which he is advised to study have either been written by members of the medical profession, or paraphrased from standard works by the older authorities on ophthalmology. This specialty, which has arisen within the lines of medical practice, cannot fairly be compared with dentistry of outside origin.

In another aspect of practical importance optometry differs from dentistry. The dentist can find full employment and an adequate income in the service of a comparatively few people. As one of them has put it, he has thirty-two patients in each mouth. Few people who take care of themselves can go more than a year without spending some time in the dentist's chair. A dentist may have plenty of work though his reputation reaches but a very few hundred people. On the other hand, the oculist may have loyal patients, and render them every service they need, and yet several years may elapse between their visits. If his work is confined strictly to the specialty of ophthalmology, he will be kept busy, and command a fair income, only when he is known by reputation to thousands. Deprive the oculist of the opportunity of becoming known through the recommendation of those who are practicing other branches of medicine, and it would be difficult for him to achieve a full practice without resorting to the methods of modern newspaper advertising. Separate ophthalmology from the other branches of medicine, and the self-praising newspaper advertiser would be placed at a distinct advantage.

But the important reason that ophthalmology should remain a recognized branch of general medicine is that the eye has most intimate relations with other important organs; and in its structure presents, readily accessible to observation, specimens of the more important general tissues of the whole body. Its lesions are samples of the changes which general morbid processes work elsewhere. They furnish the evidence of changes in other organs of the highest importance in general diagnosis. They are to be successfully combated by general remedies, diet. change of climate, internal medication, vaccine, and serum-therapy, as well as by local measures. The close connection with ophthalmology is as important to both general medicine and surgery as is that connection of each with the other, the ignoring of which, as pointed out by Clifford Allbutt and others, caused the sterile centuries of medical history.

## WHAT IS TO BE DONE?

In view of the fact that optometry is a part of medical science and art that will not and cannot be dissociated from the diagnosis and treatment of non-refractive defects and diseases of the eye; in view of the fact that it is very important that those who practice ophthalmology should be trained in general medicine, and should continue within the medical profession in close association with those who practice other branches of the healing art: what should be done with regard to the present agitation for the recognition of optometry as a separate profession?

The attempt to ignore that agitation is folly, but mere opposition, the determination to "stand-pat," is almost equally fatuous. If the optometrists are able to convince the public and the legislatures that laws are needed to protect the eyes of the people from the doings of optical fakers, mere opposition to any and every law on the subject places the medical profession in a false and indefensible position. If something is proposed as a measure to protect the public health, it is not for those who claim the right to advise on all such matters merely to oppose, while themselves suggesting nothing. It is more consistent, more public-spirited, and more effective, to endeavor so to modify legislation as to make it supplement and support the laws now enforced with reference to medical practice.

Every optometry law hereafter enacted or amended should be made to draw a sharp distinction between the license to fit glasses, and the license to practice medicine. In limiting the operations of optical fakers, it must not overlook those fakers who desire to use the word "doctor" for the purpose of confusing the public as to the training and fitness of the man who has studied only "physics" and not "physic." The simple insertion of a clause like the following, taken from the bill before the last session of the Colorado legislature, would rob optometry laws of most of their power for evil.

> "Nothing in this act shall be construed to apply to persons licensed to practice medicine in this state, or to give any person the right to attach the title M.D., surgeon, doctor, physician, oculist, ophthalmologist, eye-epecialist, doctor of refraction, doctor of ophthalmology, doctor of optometry, or any word or abbreviation to his name indicating that he is engaged in the treatment or diagnosis of diseases of, defects, or injuries

**of the human eye, or to use drugs or medicine in any form for the treatment or examination of the human eye, or to use any therapeutic measures or agencies other than glasses for the treatment of the human eye."**

No doubt such a clause would diminish the attractiveness of a license for "optometry"; and in the end might prevent the development of that pseudo-profession of which some optometrists dream. It would strictly confine the advantages of the license to the comparatively few intelligent, well-trained opticians, who wish only to work at the measurement of refractive errors and the fitting of glasses. The existence of such a class will never seriously endanger the public health, or interfere with the normal development of ophthalmology as a medical specialty.

But the most important thing the medical profession has to do is to provide for the adequate teaching of ophthalmology, including optometry, in the medical schools; to meet the needs of the practitioner of other branches of medicine, and to build up a definite class of practitioners especially trained to recognize and treat the defects and disorders of the eye. It is the failure properly to perform this duty toward the public that is responsible for the optometry question, and the larger issue of a division in the medical profession. Ophthalmology can be learned without special schools or systematic courses of teaching. Medicine and surgery were so learned by apprenticeship for hundreds of years. But the apprentice system is gone from medicine, as it is gone from the mechanic arts. As matters stand now, the graduate, after a scanty introduction to ophthalmology in the medical school, is permitted to pick up ophthalmic knowledge and skill here and there, from desultory reading, by study of chance cases, in the unsystematized clinics of postgraduate schools, from the courses of self-constituted teachers, and by a partial return to the apprentice system through assistantship in the eye clinic or private office.

This has made ophthalmologists of apt students, sufficiently conscientious to persist in the effort to obtain knowledge under difficulties. But it has also created a large number of fakers within the profession—men who pose as specialists before colleagues slightly less informed than themselves, and before the still more ignorant general public. This system, which is a lack of system, has not met the legitimate needs of the profession, or the best requirements of the public. The feeling that although a physician's license to practice gives the privilege of ophthalmic practice, it does not guarantee any fitness for such practice, has led the public to be ready to look for ophthalmic advice outside the profession as well as in it.

The establishment of the degree of Doctor of Ophthalmology, and of the course leading to that degree, in the University of Oxford points to a practical way of meeting the need of the profession and the public for some authentic evidence of fitness to practice ophthalmology. The offering of such a degree, based on requirements of graduation in medicine and evidence of special acquaintance with ophthalmic science and art, by a few of our better American universities would sweep away the false conditions, the lie in medical education, out of which the optometry question has grown. There can be no better time than to-day, and no better place than the Section on Ophthalmology of the American Medical Association for starting a demand on the part of the medical profession for the adequate teaching of ophthalmology in all well-equipped, efficient schools of medicine.

530 Metropolitan Building.

## MEMORANDA

# SHORTENING OF AN OCULAR MUSCLE BY TUCKING*

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JOLIET, ILL.

The literature of operations designed to increase the power of ocular muscles contains descriptions of more than fifty methods. All of these may have served and many of them may be still serving their originators and others in the field for which they were designed.

It must be difficult, however, for the inexperienced operator to select from this number the one which will best serve his purpose. Naturally, in making his choice he should be guided by the following considerations: First, the safety of the method; second, its effectiveness, immediate and permanent; third, the cosmetic result; fourth, ease of performance; fifth, period of convalescence.

All these operations may be divided into three classes: First, those in which a new insertion is made nearer the cornea (the advancement operation proper); second, those in which the muscle is shortened but the natural insertion preserved, as (a) the tucking operation and (b) resection of a portion; third, those which are intended both to resect and to advance. This is the method most frequently employed. The consideration of these methods is still further complicated by the fact that the muscle alone may be dealt with, or the muscle together with its overlying capsule or muscle, capsule and conjunctiva together. None of these operations is generally considered unsafe, certainly not by the originator. There is, however, a real danger in any operation in which the attempt is made to secure an artificial insertion nearer the cornea, whether the tendon is resected or not. Those methods in which the conjunctiva is advanced or resected have the further disadvantage of leaving more or less permanent disfigurement at the site of operation.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

### SUMMARY OF LITERATURE

Howe,[1] in describing advancement operations, says:

> One of the greatest difficulties in all these operations is that they require the anchorage to be made in what is usually described as the "episcleral tissue," but in reality these sutures are seldom or never in the episcleral tissue only. They are either in the conjunctiva, which is elastic and easily torn, or the needle enters the sclerotic itself. That layer near the cornea is only about 1 mm. thick. The smallest needles are usually more than 0.5 mm. thick, including the curve, and with a good thread such a needle, near its eye, measures often from 0.6 to 0.8 mm. If we use a larger needle it either does not penetrate the sclerotic or, if it does so, it is at the risk of passing through into the ciliary region.

Howe further relates the following incident:

> The danger of attempting to anchor the stitches in the episcleral tissue was once impressed on me by an accident which might occur to any one. The operator, at that time one of our leading colleagues in Berlin, was making an advancement, and on passing a small needle through this episcleral tissue the needle broke. The rough end of the part in the globe could be felt only as a point. It could not be extracted by the cilia forceps nor in any similar way. After many efforts, it was necessary to abandon the original operation, to make an incision which opened the globe, and even then the fragment could be found only after careful search for a long time.

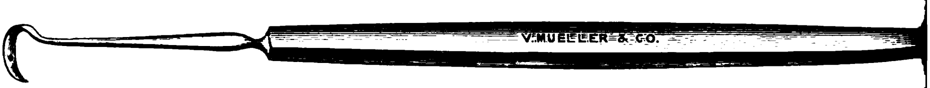

Fig. 1.—Needle for the insertion of suture in the muscle.

I have seen one case of sloughing of the sclerotic, two of perforation of the sclera, and two in which the caruncle was drawn toward the cornea.

R. Wirtz[2] reports a case of phthisis bulbi following an advancement operation, as a result of suppuration of the cornea in a patient just recovered from an attack of angina.

In a discussion of a paper by Dr. Callan[3] before the

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[1] Howe: Muscles of the Eye, ii, 350.
[2] Wirtz, R: Ztschr. f. Augenh., January, 1910.
[3] Tr. Am. Ophth. Soc., 1908, ii, Part 3.

American Ophthalmological Society, in 1908, Dr. David Webster stated that following the advancement operation he had seen one eye lost by panophthalmitis, three or four cases of corneal ulceration and on more than one occasion the operator's needle had entered the anterior chamber.

Dr. Warwick Cowgill[4] reports a case of ulcer of the cornea following an advancement operation in which there was no injury to the cornea. He attributes the ulcer to the reduction of corneal nutrition from the severing of the conjunctiva and the pressure of the anchoring suture. The eye was preserved but with a leukoma occupying one-fifth of the corneal surface and extending to its center.

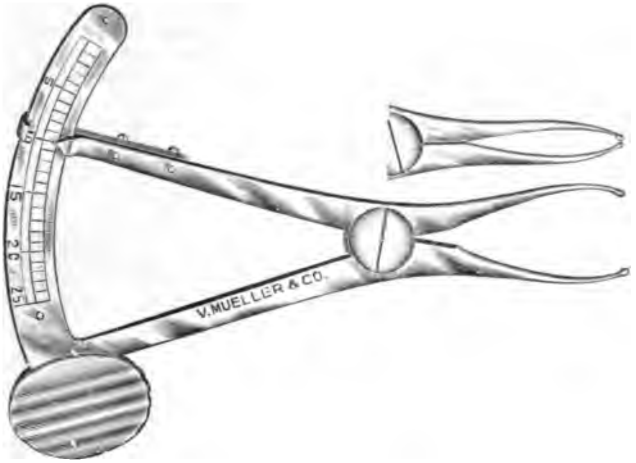

Fig. 2.—Author's strabometer.

Jackson says:[5]

> A large part of the difficulties regarding advancement operations arises from the character of the tissue dealt with. The conjunctiva and subconjunctival tissue being extremely loose, easily dragged away from their normal position and readily cut by sutures, offer a poor anchorage for supporting sutures. . . . On the other hand, the firm tissue of the sclera offers an ideal anchorage for sutures. The objection to its use is the risk of perforating the whole thickness of the sclera and opening a channel for infection to the interior of the eye-ball. . . . Even with care and in competent hands it is likely that

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[4] Cowgill, Warwick M.: Ophth. Rec., x, 3.

[5] Wood, C. A.: System of Ophthalmic Operations, i, 734.

> complete perforation of the sclera is not very rare. Cogan, passing sutures in pig's eyes, found the sclera perforated in half of them.

H. D. Noyes,[6] referring to deep anchorage at cornea sclera junction, wrote: "I have seen the needle points sometimes appear in the anterior chamber."

My chief criticism of the so-called advancement operation, however, is the insecurity afforded the sutures in muscle and in sclera. Beard says:[7]

> A great deal has been said about the advantage of this or that advancement procedure in that the arrangement of the suture or sutures in the tendon tends to prevent its cutting out. From an abundant experience with his own method both as performer and onlooker, the writer has long since come to the conclusion that it is far easier for the average surgeon to obtain a firm hold for the thread in capsule and tendon than in the globe.

The reason serious accidents do not follow the advancement operation oftener is that the operator, erring on the side of safety, prefers insufficient anchorage to perforation of the sclera.

The authors of the various advancement operations claim ease of performance, and it is difficult for one to judge by description alone as to this point. None of them is as simple as tenotomy and for this reason one is often tempted to cut the stronger muscle when his better judgment tells him that the weaker should be strengthened. One of the strongest claims for the advancement operation has been that the muscle takes its attachment alone from the new insertion, thereby increasing its power very much. Müller[8] has pointed out that the only effect of advancement is to shorten the muscle, for the advanced muscle probably grows fast to the bulb along the entire interval between the side of the wound and that of the old insertion and does not unwrap itself from the globe even in extreme rotation. Hence the advanced muscle is practically inserted at the site of the original tendinous insertion.

Froelich says:[9]

> If the end of an ocular muscle is transplanted over its natural insertion toward the cornea, the inner surface of the advanced muscle grows fast to the outer surface of the sclera.

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[6] Noyes, H. D.: Text-Book on Disease of the Eye, p. 172.

[7] Beard: Ophthalmic Surgery, p. 203.

[8] Müller: Klin. Monatsbl. f. Augenh., xxxi, 127.

[9] Froelich: Arch. Ophth., xxxiv, 622.

This was shown in the following case: A young man needed a readvancement of his right external rectus which had been advanced some years before. The conjunctiva was undermined outward from the scar at the margin of the cornea, so as to lay bare the dark red muscle which had to be separated from the sclera with scissors. The attachment was firm and extended exactly to the physiologic insertion, beyond which the relations between the muscle and sclera were normal.

In the same article, Froelich further says:

The usual explanation of the effect of an advancement is that because the advanced muscle girdles the globe to an extent increased by the distance between the original insertion and the margin of the cornea, its efficiency is increased so as to enable it to produce greater motility of the eye-ball. But this is not the true explanation as I demonstrated in the following way: During the advancement of an external rectus of a leukomatous eye, I measured the distance of the insertion from the corneal margin with a pair of compasses. Frequent measurements of the excursions of the eye were made during the next six weeks, and at the end of that time I made a curved incision 1 cm. long, parallel to the line of insertion, down to the sclera, at the distance from the cornea previously determined. This incision completely isolated the transplanted end of the muscle from the rest, and yet the efficiency of the muscle was not in the least impaired. This showed that after the operation the anatomic insertion still formed the point of termination and insertion of the muscle which had been shortened by the portion advanced and thereby rendered more able.

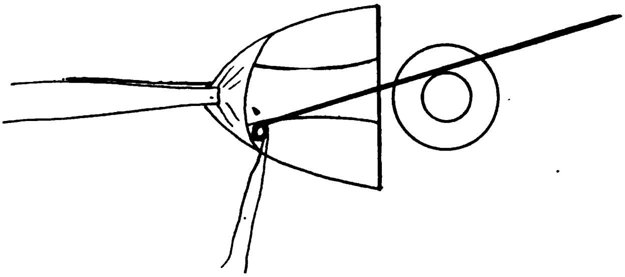

Fig. 3.—Showing method of inserting suture in the muscle border.

Jackson says:[10]

It has been assumed that an advancement operation causes the tendon of insertion to form a new attachment to the eyeball at its anterior extremity, and to act on this attachment as it did on the original insertion. It is on this assumption

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[10] Wood, C. A.: System of Ophthalmic Operations, i, 736.

**that the beautiful demonstration of the superiority of advancement over tenotomy of Landolt and his followers has been based. But the writer's experience in secondary operations agrees with that of others to indicate that the advanced tendon does not become attached to the eye-ball simply by its anterior extremity, but rather that the attachment extends at least as far back as the original insertion of the muscle and sometimes farther.**

It is evident, then, that other operators have recognized these disadvantages as indicated by the history of the tucking operation. It is supposed to be the result or rather the outgrowth of the operation of de Wecker who first advanced the muscle without severing the tendon. Since then various operations have been described by Lagleyze,[11] Savage,[12] Suffa,[13] Bourgeois,[14] Maxwell,[15] Clark,[16] Bruns,[17] Green,[18] Todd,[19] Levinsohn,[20] Briggs,[21] Valk,[22] Colburn,[23] Suker.[24]

These methods vary in the manner of making the tuck, insertion of the sutures and the use of special instruments or tuckers. Some make the fold on the distal side of the tendon insertion and some on the proximal side. These latter do not differ in principle from the de Wecker method. It being necessary to suture to the sclera, the only advantage over advancement with severance of the tendon is that if the suture tear out the result can be no worse than at first.

## TECHNIC

My experience with the tucking operation began about nine years ago at the Illinois Eye and Ear Infirmary. My favorite advancement operations at that time were of Worth and Wooten and, in what I will call the simple tucking operation, I have retained the important features of each. At that time, Dr. Henry Boettcher suggested trying Todd's new tucker and we performed a number of operations according to this method, finally doing away with the tucker. At the March, 1910, meeting of the Chicago Ophthamological Society,[25] I showed six patients who had been operated on for strabismus, and described the operation. Since then, I have still further demonstrated the fitness of the operation, and have derived considerable satisfaction by the use of the special needle shown in Figure 1, suggested to me by Dr Tivnan, and by a pair of compasses (Fig. 2), by the use of which an approximate idea of the relationship between the amount of tucking and angle of correction may be obtained. This strabometer is graduated in millimeters. On the back of the scale is a cog which slips into a depression at every millimeter and rests there securely. This arrangement is the same as that on the ophthalmoscope and was suggested by Dr. Schneider of Elgin. Also, during the year I succeeded in correcting divergence of 65 degrees by double tuckings of the interni with tenotomies of the externi, securing binocular single vision.[26] This patient was shown at the December, 1910, meeting of the Chicago Ophthamological Society.[27]

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[11] Lagleyze: Arch. d'opht., 1892, xxii, 668.
[12] Savage: Ophth. Rec., March, 1893.
[13] Suffa: Arch. f. Ophth., 1910, xxxviii, 254.
[14] Bourgeois: Bull. et mém. de la Soc. franc. d'ophth., 1906.
[15] Maxwell: Brit. Med. Jour., 1896, ii, 819.
[16] Clark: Tr. Sec. on Ophth., Am. Med. Assn., 1900, p. 164.
[17] Bruns: Ophth. Rec., 1904, p. 267.
[18] Green: Ophth. Rec., 1899, p. 462.
[19] Todd: Ophth. Rec., 1902, p. 73.
[20] Levinsohn: Klin. Monatsbl. f. Augenh., September, 1909, p. 299.
[21] Briggs: Tr. Am. Acad. Ophth. and Oto. Laryngol., 1908, p. 212.
[22] Valk: Post-Graduate, July, 1895.
[23] Colburn: Ophth. Rec., April, 1902.
[24] Suker: Am. Jour. Ophth., May, 1904.

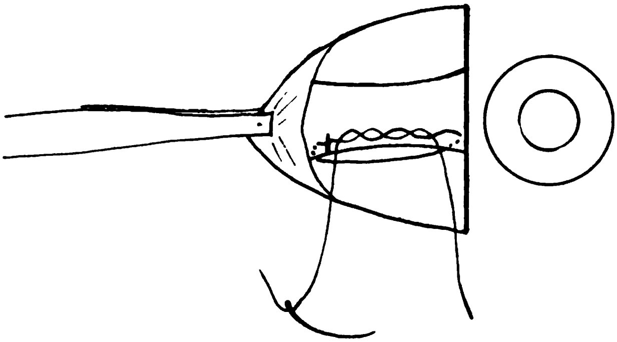

Fig. 4.—Illustrating method of tying the Worth suture.

### TECHNIC OF OPERATION

The operation is now performed as follows: Local anesthesia is secured by the injection of from 20 to 30 minims of a 1 per cent. solution cocain with 5 minims of

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[25] Woodruff: Jour. Ophth. and Oto. Laryngol., April, 1910, p. 1.
[26] Woodruff: Jour. Ophth. and Oto. Laryngol., March, 1911.
[27] Worth: Ann. Ophth., July, 1902, p. 377.

Epinephrin solution added beneath the conjunctiva over the site of the muscle. The resultant swelling is massaged until it has almost disappeared. A vertical incision is made through the conjunctiva only, over the site of the tendon insertion about 15 mm. in length. The flap is dissected up from the underlying capsule as far back as 15 or 20 mm. While this flap is held out of the way by an assistant, the capsule is incised along the upper border of the tendon as far as the conjunctival dissections. A similar incision is made along the lower muscle border. Formalized pyoktanin catgut No. 00, is inserted into the muscle by the use of the needles shown in the cut; these needles resemble aneurysm needles on a small scale. It is simply necessary to pass the needle point through the muscle border (Fig. 3) about one-quarter of its width drawing the thread through with the forceps. The suture is firmly tied in a double knot and one end threaded through an ordinary curved needle is again passed through the muscle posterior to the knot from without, inward, according to Worth[27] (Fig. 4). This suture which lies next to the sclera is passed through the tendon at its insertion from below upward or rather forward. A similar suture is placed in the upper muscle border. When these two sutures are tied, the tuck is formed in muscle and capsule, and the muscle correspondingly shortened (Fig. 5). The conjunctival edges are then brought together with silk. The eye operated on alone is bandaged.

### RESULTS

1. The highest degrees of strabismus may be corrected by tuckings with tenotomies of the opposing muscles.
2. Torsion is prevented by measuring the distance from the tendon insertion back to the point of entrance in the muscle and making both upper and lower correspond.
3. The method of tying the suture in the muscle prevents its splitting the muscle fibers while traction is made on it and when tied to the tendon insertion it is a physical impossibility for the muscle to become loosened as the traction is at right angles to the direction of the muscle fibers.
4. By disturbing the capsule so little considerable swelling is avoided, Wooten,[28] but by including the overlying capsule, somewhat greater effect is produced.

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[28] Wooten: Ann. Ophth., July, 1902, p. 431.

5. By not sacrificing any portion of conjunctiva or advancing it, deformity and unsightly scar tissue are avoided.

6. A tucking of a given amount of muscle will result in not more than half as much correction in the position of the eye.

In a convergent strabismus of 10 mm. it is well to tuck 15 mm. of muscle and tenotomize the rectus internus leaving a slight undercorrection. For anatomic reasons a 15 mm. tuck is about as much as one is able to make. Then, too, the externus itself is about 50 mm. in length and a shortening of a greater proportion might easily impair its power of contraction. Motais has shown that the contraction of a rectus muscle necessary for its full physiologic movement is about one-half its length.

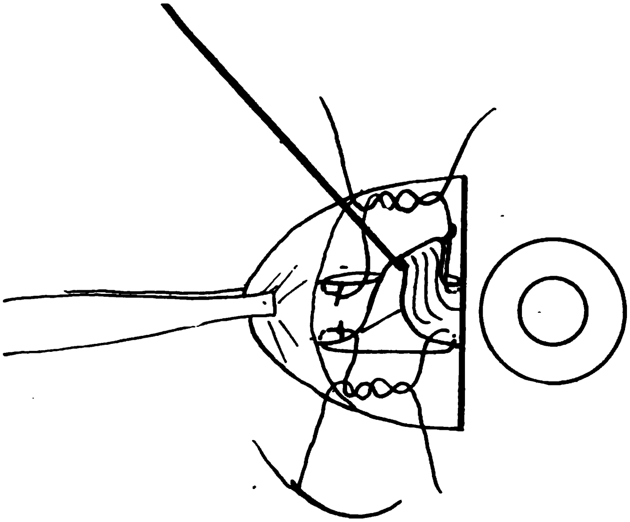

Fig. 5.—Portion of the muscle held up on a strabismus hook which becomes the tuck when the sutures are tied.

I feel that I can recommend this operation to my colleagues as being absolutely safe, permanently effective, devoid of any permanent noticeable deformity, easy to perform under local anesthesia in any one over 12 years of age. The period of convalescence need not be longer than one week.

Woodruff Building.

# A NEW METHOD IN OPERATING FOR ECTROPION *

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NEW YORK

The object of the present paper is to present a new principle in operating for ectropion, which is applicable, either singly or in conjunction with other methods, in all forms of atonic ectropion, and in all cases of cicatricial ectropion in which the tarsus itself is not destroyed.

I say a new principle: it is new so far as I have been able to find out by consulting the literature at my command and by talking with my confrères. However, when we consider that Antillus devised an operation for this deformity fifteen centuries ago, and that operations and modifications innumerable have been practiced since then, it would be small wonder if this principle had not been hit on before.

The principle consists in shortening the tarsus at the temporal extremity, and then *attaching this extremity to the periosteum* of the temporal border of the orbit, on a level slightly above the lid commissure if the lower lid is affected, and below the commissure if the upper lid is in fault. The operation is performed as follows:

## ATONIC ECTROPION

1. A canthotomy is performed extending to and slightly beyond the orbital margin, exposing the periosteum.
2. The edge of the lid including the cilia is pared off to a slightly greater extent than the piece of tarsus to be removed.
3. The skin and orbicular muscle are separated from the external canthal ligament and the outer end of the tarsus.
4. A triangular piece of the tarsus, more or less extensive according to the extent of the ectropion and lengthening of the lid border, is excised. In case of redundancy of the skin,

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a triangular piece of skin equal to that of the removed tarsus is removed.

5. A No. 6 silk suture is placed through the outer extremity of the shortened tarsus near the margin, the needle being passed from without inward; then the needle is carried through the periosteum of the rim of the orbit (slightly above the commissure if the lower lid is being operated on, and slightly below if the upper lid is being corrected); the needle is brought out and continued through the skin from the under surface outward. The suture is tied in a single knot. If enough tarsus has not been removed to correct the deformity, the suture is loosened, a little more of the tarsus excised, and the anchoring suture reapplied as before.

The skin wound is closed with fine silk sutures. A dry gauze dressing is put on and a bandage applied. The skin sutures should be removed on the third or fourth day, while the anchoring suture, which can be reached on the skin surface, where it is brought out on purpose, should not be removed until the seventh day.

In cases of excessive atonic ectropion and in many cases of cicatrical ectropion, instead of a piece of skin being cut from the lid itself, a triangular piece may be removed beyond the external canthus, as in the Dieffenbach-Szymanowski operation. Then the undermined skin from the lid is slid over and sewed in position, just as in that operation. It supports the cartilage which has been anchored to the periosteum, and the combined operation is very effective and is advised in marked cases of ectropion.

## CICATRICIAL ECTROPION

This principle of anchoring the tarsus to the periosteum applies in many cases of cicatricial ectropion; in fact in most of the cases in which the tarsus itself has not been destroyed. Even when the skin must be replaced by a flap or a skin graft, if the tarsus is first freed and the outer extremity anchored to the periosteum of the rim of the orbit, the result, as a rule, is satisfactory. The first case in which I used this method was one of very extensive cicatricial ectropion; one that had had six operations, and which I report, as the result was most satisfactory and has remained so for fifteen months.

*History.*—Helen C., aged 17, in good general health, fell on a stove when 9 months of age and burned the left side of face and forehead, which caused marked cicatricial ectropion of the upper and lower lids.

*Examination.*—There was mechanical ptosis of the upper lid, due to a thick and broad pedicle flap from the forehead, with slight entropion of this thick lid at the outer extremity;

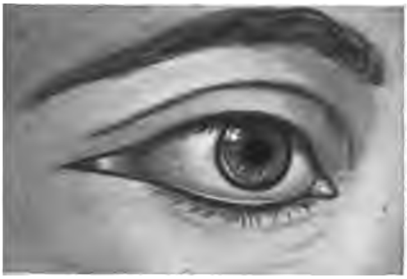

**Fig. 1.**—This and the following five figures show the author'soperation for ectropion. Canthotomy, the first step.

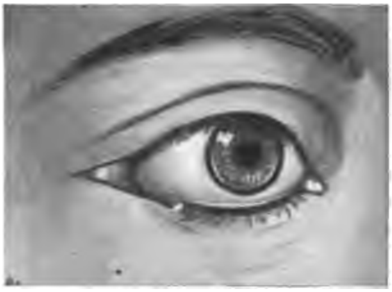

**Fig. 2.**—Tarsorrhaphy.

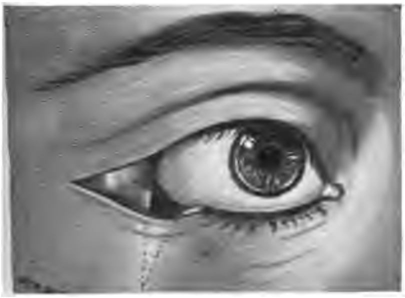

**Fig. 3.**—Triangular piece of tarsus removed.

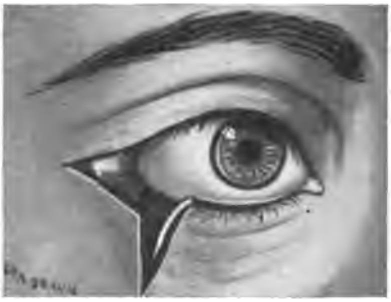

**Fig. 4.**—Triangular piece of skin removed.

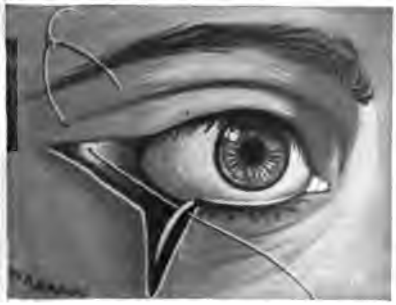

**Fig. 5.**—Insertion of anchoring suture.

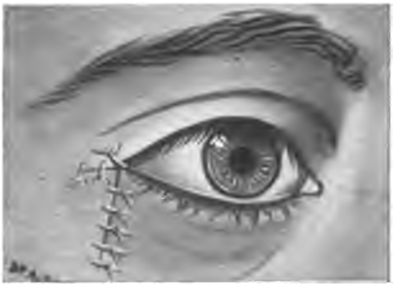

**Fig. 6.**—Completed operation.

complete ectropion of the lower lid, scar tissue binding the lid to the periosteum. There was extensive scar tissue extending an inch from the external canthus, up and down and toward the temple. The eye diverged 20 degrees and there was constant epiphora.

*Operation and Results.*—Dec. 1, 1909, under ether anesthesia, I operated as follows: Beginning at the external canthus an incision was made outward and slightly upward beyond the rim of the orbit, exposing the periosteum. A second incision, double the length of the first, extending directly downward from the external canthus was next made, and the ends of these two incisions joined by a third, as in the Szymanowski method. This triangular piece of scar tissue was excised. The outer edge of the lid, including the cilia, was pared to the extent of about one-fourth inch. Next, the skin and muscle were undermined, separating them from the periosteum far down on the cheek and from the tarsus up to its free border, but not from the free border. Incidentally the tarsus itself was freed from the periosteum of the cheek. A triangular piece of the tarsus, one-fifth inch long on the free border, was excised, a No. 6 silk suture was placed as described in step 5. On the fourth day the skin sutures were removed and on the seventh the deep anchoring suture. The result, to tell the truth, was much better than expected. The ectropion was completely relieved and has remained so to date, and there is no epiphora at all.

A few weeks afterward, I removed a strip from the upper lid, relieving the ptosis almost entirely, and a little later I performed an advancement on the left internal rectus muscle with tenotomy of the left external rectus, getting a perfect cosmetic result, though the patient has not single binocular vision, the amblyopia being very pronounced in the left eye.

In the few cases of atonic ectropion in which I have tried this operation in its simplest form, that is, by removing a small piece of tarsus and anchoring the outer extremity to the periosteum, removing no skin and only a few cilia, the result has proved entirely satisfactory in every case.

50 West Thirty-Seventh Street.

## MEMORANDA

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# PLASTIC SURGERY OF THE EYELIDS, USING THE WOLFFE GRAFT *

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ATLANTA, GA.

Plastic surgery has always impressed me as being the height of all surgical skill. For obvious reasons plastic surgery about the face, about the eyelids particularly, not only requires good judgment on the part of the operator, but his technic is put to the severest test.

The operation for the restoration of the lids and of the cul-de-sacs in contracted orbits, in the complete ectropions, especially from burns, and many other conditions, makes the deep cutaneous graft of Wolffe the ideal one to be used in such cases. My experience in twenty-five cases in which this cutaneous graft was applied has been so satisfactory and the method employed is so simple that I present the subject with the hope that it will be of interest. No claim is made to originality in the operation to be described, for my ideas were obtained from observing other operators.

There are two important steps to be remembered: First, the surface to be grafted must be denuded and must have sufficient blood-supply to nourish a healthy graft; second, a graft must be obtained of sufficient size to more than cover the area.

The ideal case is a complete ectropion of the upper or lower lid, and the one I now report, in which the result was most brilliant, was that of a farmer who, eighteen years before the first operation, had fallen into an open fire, burning the left side of his face and forehead. In time there was a complete distortion of that side of the face from the cicatricial contraction, and the ciliary margin of his upper eyelid occupied the position of his eyebrow, which had been destroyed, and the margin of

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the lower eyelid was pulled well down on the cheek. The conjunctival surfaces of the lids were completely everted and exposed, and from prolonged exposure they had become much thickened, resembling a velvety type of

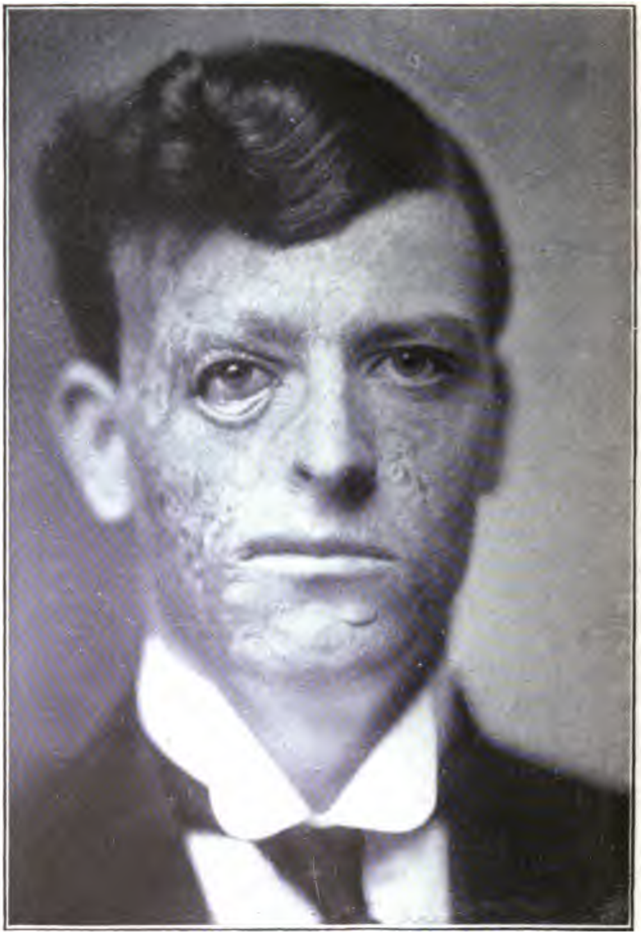

Fig. 1.—Ectropion of upper and lower lids of twenty years' duration, from burn of face

trachoma. The picture he presented was horrible. Fortunately the man had excellent vision, the globe in no way being injured. Two operations, at six months' interval, were required, the upper eyelid being first operated on.

After cleansing the face with ivory soap and water, then irrigating with a normal saline solution, I made a superficial incision above the ciliary margin, then little by little I undermined the whole lid, as one would in an ordinary integument dissection, preserving the muscular

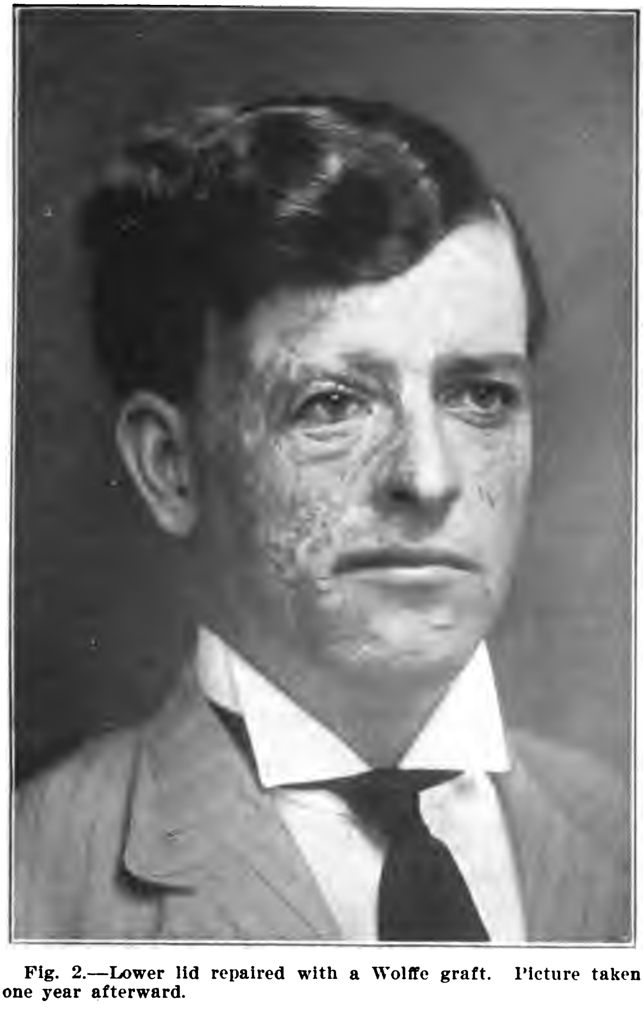

Fig. 2.—Lower lid repaired with a Wolffe graft. Picture taken one year afterward.

structure. This flap of the lid, when folded down over the globe, nearly covers the entire exposed conjunctival surface of the lower lid. Bleeding is of no consequence, but should this be troublesome a small artery clamp applied for a few moments would control the annoying vessel. After this part of the operation is completed, and the operator is sure that the eyelid will remain in place from its own weight, a pad saturated with hot saline solution is applied and gentle pressure made by an assistant, while the graft is obtained from the inner surface of the arm, forearm or thigh. I have never obtained a graft except from the inner arm, and in one case I removed, at intervals, five grafts from the two arms. The left arm is generally prepared, after the usual manner of shaving, cleansing with green soap, bichlorid of mercury and a saline dressing applied some hours before the operation, so that the graft will be as little irritated as possible at the time of the operation. A nurse or an attendant holds the arm out from the body by the hand, and the arm is wrapped with sterile towels.

Some idea should be had as to the size of the place to be covered, and I usually measure it roughly with the handle of my scalpel, then make the graft at least one-third larger, allowing for shrinkage and a loose fit. An oval graft is advisable, as the wound of the arm which is to be subsequently closed leaves only a linear scar. I usually mark off on the skin of the arm with the knife four small lines indicating the size of the graft, then connect them with a superficial incision, and as the graft is removed the incision is deepened, extending down to the fatty layer. A pair of tissue forceps and a Desmarres lid scarifier are the only instruments necessary in this step of the operation.

The graft should not contain any of the areolar or fatty tissue, and after its removal, with a pair of scissors curved on the flat, the raw surface should be trimmed of all the loose and unnecessary tissue; it is then placed in a towel wet with warm saline solution and is ready to be applied to the raw surface previously made.

The arm wound can be repaired by an assistant, or later by the operator. This operation simply consists in undermining the edges sufficiently to bring them together without tension. Two or three silkworm gut retention sutures are best used, followed by a continuous silk suture of coaptation; a dry dressing is applied and the wound is not disturbed until the fourth day. The nurse should watch for swelling of the hand for several hours after the operation; if present, this would indicate a restricted blood-supply from too tight a bandage or tension of the wound.

The new graft should be applied immediately to the raw surface for its nourishment, but all bleeding of the wound must first be controlled, lest a blood clot subsequently forming beneath the graft delay healing or form a good nidus for infection. I use a strabismus hook to smooth the graft into place, and it is remarkable how an irregular surface can be beautifully and evenly covered. Small sutures of No. 5 black silk are used to hold the graft in place. They are first placed at four equidistant points, to even the graft, then the space between is filled in with as few as possible to hold the graft steady. If the graft is too large, it can be trimmed after it is in place; but it is better to have a too large, loose fitting graft than a too small, tight one. The subsequent contraction may spoil a beautiful operation.

A dry dressing is applied, made up of several layers of gauze to absorb the secretion, then loose gauze on top. The first dressing should not be made sooner than the fourth day, then the adherent pieces of layer gauze should be moistened in order not to pull apart the wound. The sutures can be removed, or at least a part of them, for I have found that delayed removal may cause a slight infection, which may destroy the graft. Sterile petrolatum should be gently smeared on the wound, and a tight dry gauze dressing applied. At intervals of two or three days the dressings should be changed until the superficial epithelium of the graft commences to slough, then the wound should be cleansed daily. The slough may be frequently trimmed. In about three weeks the patient can go without a bandage or other protection, but for many months later it is well to have the patient massage the new lid daily with petrolatum, so that it may be kept soft.

In my second case of ectropion of the lower lid there was a slight recurrence two months afterward, but daily massage entirely corrected the condition. The method of restoration of the cul-de-sac by using the Wolffe graft is described in the excellent monograph of Weeks.[1] While the major points of that operation are similar to the method just described, some niceties in technic and the application of sutures and the use of the dental rubber plate for the holding of the graft are more thoroughly explained.

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[1] Tenth Congress of Ophthalmology, Lucerne, 1904.

While the operation that I have just described applies best to flat surfaces to be grafted, chiefly ectropions, I have recently used it with success in two nose cases in which the alæ had been destroyed by burns, and in a case of burn in which the upper lip had been pulled high up on the cheek from the scar contraction. The vermilion border was undermined, the scar in part being dissected out and a very loose graft applied. The appearance was much improved.

So far I have not had an infection or a complete failure. The nearest approach to failure occurred in a severe case of ectropion of the lower lid referred to me by Dr. W. P. Nicolson. The patient had had a mole removed from his cheek years ago by the application of some caustic paste, which had caused so much tissue necrosis that most of the soft tissue of the cheek on one side had been destroyed, exposing parts of the upper and lower jaws. Dr. Nicolson restored the cheek by a pedunculated graft removed from the neck, and referred the patient to me to correct the lid deformity. There was little sound surface for the graft, except the deflected ectropic lid, and the slough that followed only delayed epidermization.

672 Peachtree Street.

# MEMORANDA

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# ANOTHER CASE OF CHLOROMA*

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The bond between the special fields in medicine is becoming closer every year. The object of this paper is to record a case of chloroma and refer to a report of another already published, thus evidencing the broad relationship between ophthalmology and general medicine. Because of the rarity of the lesion and the frequency of ocular involvement the subject seems of sufficient interest to warrant the citation of these cases with a list of reported experiences. While the true significance of chloroma is not thoroughly understood, and although its general classification is in doubt, it is probably a systemic condition with local symptoms. This last statement may not pass unchallenged, but it is admittedly the opinion of those pathologists and hematologists whose experience with this disease gives their authority most weight.

**Case 1.**—J. L., male, aged 8, of foreign birth, was first seen Dec. 3, 1906.[1]

*General Examination.*—Extremely emaciated, skin distinctly yellowish, mucous membranes anemic; the right eye proptosed 11 mm., both lids somewhat discolored with bluish tinge but freely movable, palpebral veins enlarged; appearance of a growth in the upper part of the orbit due to slight edema. The lower orbital ridge was definitely outlined, but a firm, freely movable tumor mass 12 mm. wide extended from the outer to the inner canthus. This mass was free from skin, conjunctiva and bone. Its posterior border was not distinctly palpable. Bulbar and palpebral conjunctiva negative, except for two 5 mm. irregular, subjunctival hemorrhages near the limbus over the external rectus. Cornea clear, anterior chamber negative, iris normal, pupil 4 mm., active, media clear. Marked optic neuritis was present with complete obliteration of the disk outlines; white exudates along the larger vessels, but in no other part of the retina; no retinal hemorrhages; vision-fingers at 6 feet; complete ophthalmoplegia externa. The left eye was extremely prominent, extending 20 mm. beyond the superior orbital ridge. The temporal, frontal, angular and supra-orbital veins were enormously distended and tortuous, with dark-blue lids. Projecting beyond the upper orbital margin was a firm growth not adherent to the surrounding structure. It seemed to extend deep into the orbit and was palpable from the supra-orbital foramen outward beyond the outer canthus. Closure of the lids was impossible; conjunctiva markedly chemotic, with numerous ecchymoses; cornea clear above, the lower half covered with yellowish masses of dried exudate; anterior chamber and iris negative. Pupil 5.5 mm., but does not react to light or accommodation. Deep in the vitreous a yellowish-grey reflex was obtained, but no fundus details; no light perception; complete ophthalmoplegia.

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[1] Merrill and Bedell: Chloroma, with Special Reference to the Ocular Symptoms, New York State Jour. Med., October, 1907.

*Blood Examination.*—Dr. Rooney's examination of the blood Nov. 26, 1906, showed:

| | |
|---|---|
| Reds | 1,596,000 |
| Whites | 41,200 |
| Hemoglobin | 30.0 per cent. |
| Color index | 1.+ per cent. |

Differential count of 400 whites:

| | |
|---|---|
| Polymorphonuclears | 37.9 per cent. |
| Large mononuclears | 11.0 per cent. |
| Small mononuclears | 12.0 per cent. |
| Eosinophils | 3.1 per cent. |
| Myelocytes, neutrophilic | 32.8 per cent. |
| Myelocytes, eosinophilic | 7.2 per cent. |

Poikilocytosis; many micro- and macrocytes; many degenerated whites.

Dr. A. T. Laird's blood examination, Dec. 8, 1906, showed:

| | |
|---|---|
| Reds | 1,410,000 |
| Whites | 79,600 |
| Hemoglobin | 30.0 per cent. |

Differential count of 6,000 leukocytes:

| | |
|---|---|
| Polynuclears | 16.2 per cent. |
| Large mononuclears | 5.3 per cent. |
| Large lymphocytes | 15.3 per cent. |
| Transitionals | 0.8 per cent. |
| Eosinophils | 0.7 per cent. |
| Small lymphocytes | 33.0 per cent. |
| Neutrophil myelocytes | 28.2 per cent. |
| Eosinophil myelocytes | 0.5 per cent. |

Five nucleated red cells were noted; three were normoblasts, two were larger than normoblasts and were classed as megaloblasts. A number of degenerated leukocytes were seen, but no record of them was made.

*Operation.*—The patient was given chloroform and a 3 cm. incision was made over the outer half of the superior orbital ridge. The entire orbit was filled with a greenish mass of almost cartilaginous consistence, having a definite capsule in parts, no large blood-vessels and not palpably adherent to the periosteum. The optic nerve could be definitely outlined, but the ocular muscles could not be distinguished. Palpation to the apex of the orbit proved that the entire content consisted of this pale green tumor.

The tissue removed was sent to Dr. R. M. Pearce of the Bender Laboratory, who reported as follows:

*Pathology.*—Tissue from the left orbit. Material consists of three small pieces of tissue, the largest about 0.5 cm. in diameter. These are irregular in shape; in part firm, of light greenish color, and in part soft and pink in color. On section they show a uniform smooth surface, firm in the green portions and soft in the pink portions.

*Histology.*—The centers of the nodules are composed of closely arranged cells of the type of the larger lymphoid cell. These have a deeply but slightly irregularly staining nucleus and a small ring of eosin-staining protoplasm. As a rule, they are round or slightly oval in shape, but occasionally are irregular, resembling in a general way the plasma cell. No multinucleated cells are seen. Toward the periphery are single fat spaces which have resisted the general infiltration. These spaces are more abundant at the extreme periphery, but the tissue between is extensively invaded by the new cells. Only here and there are many small areas of uninvolved fat tissue seen. Here also are areas apparently at the point of periosteal attachment, with considerable fibrous tissue infiltrated with lymphoid cells, more or less hyaline in character. In the center of the nodules there is but a faint reticulum of indefinite arrangement. Few blood-vessels are seen; polymorphonuclear leukocytes are not present in appreciable numbers.

*Subsequent Course.*—The patient's condition grew progressively worse from date of admission. Emaciation was extreme. Proptosis of both eyes became more marked. Tumor mass on the right side increased 2 mm. in width and the eyelid became more darkly congested, and the vessels tortuous and prominent. Vision was almost totally lost, but owing to the patient's mental state, could not be determined definitely. Pupil 6 mm., not reacting. No increase in the retinal changes. The left eye began to show signs of lagophthalmic keratitis. The lower half of the cornea was infiltrated and the outer layers eroded. No fundus was visible; no decrease in the chemosis; veins more enlarged.

December 10, at 11 a. m., the child had a convulsion which started with general muscular tremor of the right side, followed by marked spasm with rotation of body toward the left. This side was entirely paralyzed, respirations stertorous, patient unconscious, but at times seemed semi-conscious. Further observation of his condition prior to death on the same day was prevented by his removal from the hospital. Autopsy was not permitted.

The second case was under observation nine days, during which time complete examinations were made by Dr. Wolbach.

**Case 2.—Mrs. N. G., aged 18, married, a Russian by birth, was admitted to the Albany Hospital on the service of Dr. G. Emory Lochner, April 7, 1909. Family and personal history negative. Patient menstruated from 13 years of age until one week after marriage, July, 1908. One month prior to admission she first noticed many small, hard, insensitive lumps in each breast, which she believed had not increased in size. March 8, 1909, she had "pink eye," from which she seemingly recovered; on March 27, her eyes began to bulge, causing pain and attacks of blindness.**

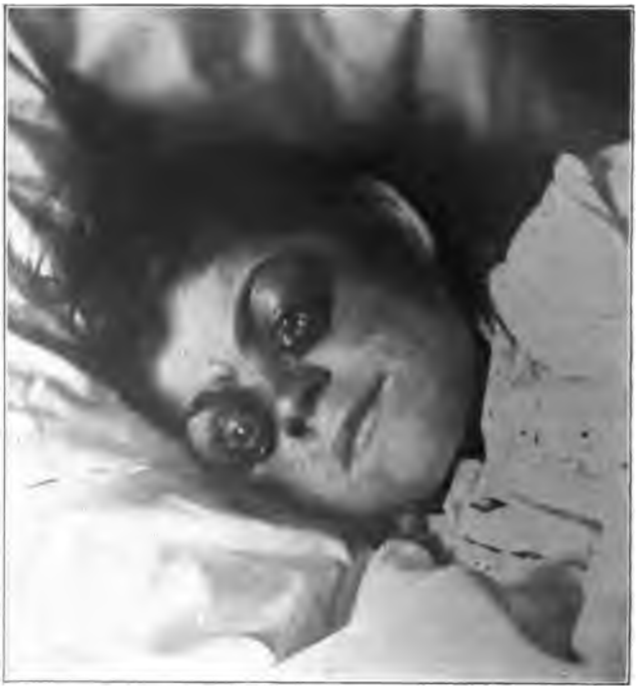

Fig. 1.—Case 2.

*General Examination.*—The patient was an undersized, delicately built, poorly nourished woman. The skin had a peculiar satiny texture and was of grayish-yellow color. Several nodules about 2 cm. in diameter, which were attached to muscle and bone, were palpable in each breast and along the sternum. The axillary and inguinal glands were enlarged. The woman was eight months' pregnant; a systolic murmur was audible over the whole precordium and not transmitted. These were the only departures from an otherwise negative physical examination, except for the ocular lesions and blood changes.

April 8, the right eye proptosed 20 mm., immobile. The upper lid showed great dilatation of the blood-vessels, and although it covered one-third of the cornea, it might be retracted, while the lower lid was covered with chemotic conjunctiva. A definitely palpable tumor mass 20 by 12 by 8 mm. was outlined over the lacrimal gland, entirely in the lid, with no deep attachment. The superior two-thirds of the cornea was clear, but the lower third showed a dense interstitial and superficial haze. Pupil 1.5 mm. Vision-fingers, 3 feet.

Left eye proptosed 18 mm.; small nodule in the upper lid at the outer side; cornea hazy below, pupil 4.5 mm. stationary. Vision, fingers at 3 feet. A large mass of grayish appearance pressed the retina forward, and, except for an occasional vessel, obscured fundus detail.

*Blood Examination.*—Blood examination made by Dr. James F. Rooney, April 8, showed:

| | |
|---|---|
| Reds | 3,150,000 |
| Whites | 36,500 |
| Hemoglobin | 55.0 per cent. |

Differential count of 300 white cells:

| | |
|---|---|
| Polynuclears | 15.8 per cent. |
| Lymphoblasts | 37.2 per cent. |
| Small mononuclears | 37.0 per cent. |
| Large mononuclears, transitionals | 9.0 per cent. |
| Eosinophils | 0.8 per cent. |
| Mast cells | 0.2 per cent. |

Five normoblasts seen in counting 300 leukocytes.

Four days later, April 12, the condition was much as shown in Figure 1. The right eye bulged forward 25 mm., with intense engorgement of veins of lids and temporal region.

The tumor mass was now 25 by 12.5 mm., axis 60 degrees, not adherent to the skin or periosteum, although extending deep in the orbit. The outer part of the lower lid was filled by an oval, unattached growth 18 by 9 mm., axis 120 degrees.

The lower half of the eye-ball, including the entire cornea, was exposed.

The conjunctiva was chemotic, with many small ecchymoses, and a small hypopyon in the anterior chamber. Tension normal; vision = L. P.; pupil 2 mm., stationary; extremely limited motion of the globe.

Left eye proptosed 22 mm., with many irregular nodular growths in the upper, outer two-thirds of the orbit, close to the superior wall, but not adherent; upper half of the cornea covered by a vein-filled upper lid, the lower half rough, beginning to slough; eye-ball stationary; bulbar conjunctiva decidedly congested; the interior of the eye unchanged; anterior chamber negative. Pupil 45 mm.; faint reaction to light; tension normal.

Dr. Rooney's blood examination, April 12, showed:

| | |
|---|---|
| Reds | 2,200,000 |
| Whites | 52,000 |

Differential count of 300 whites:

| | |
|---|---|
| Lymphoblasts | 45.2 per cent. |
| Polynuclears | 10.6 per cent. |
| Small mononuclears | 19.2 per cent. |
| Large mononuclears and transitionals | 8.0 per cent. |
| Eosinophils | 3.0 per cent. |
| Myelocytes | 0.7 per cent. |
| Degenerated | 1.0 per cent. |

Six normoblasts, one megaloblast.

April 16, the patient died, after premature delivery of a dead child. As in many cases, persistent uncontrollable nasal hemorrhage was present for several days before death.

The following is the autopsy report of Dr. Wolbach, held April 16, 1909:

*Autopsy.*—Portion of breast tissue excised three hours post mortem shows glandular and fat tissue invaded by many tumor nodules from a few mm. up to 3 cm. in diameter. These nodules are of firm consistence and vary in color from a light green, the color of cooked peas, to an olive green.

Autopsy limited to chest and abdomen and done under a very short time limit.

Body is that of a short-statured, slightly built white woman. There is extreme double exophthalmos and both eye-balls are reddened and covered with puriform material and crusts. Breasts are large and nodular. The areolæ are deeply pigmented. Mid-line of the abdomen is deeply pigmented. Rigor mortis is complete. Marked post-mortem lividity of dependent parts. No edema.

Peritoneal Cavity: Peritoneum is smooth, moist, glistening. Appendix normal. Mesenteric lymph nodes not enlarged.

Chest: Under surface of the sternum is covered with many green tumor nodules which are situated on the surface and beneath the periosteum. The intercostal muscles on both sides for a distance of several cm. are invaded and replaced by tumor tissue which completely surrounds the costal cartilages of the upper five or six ribs.

Pleural and pericardial cavities negative.

Heart: Normal in size. Myocardium is of good consistency. Valves and endocardium are normal. There are three tumor nodules in the auricles of the heart situated as follows: one in the posterior wall of the left auricle near the interauricular septum. This nodule is olive green in color and measures 2 by 1.5 cm. In the interauricular septum just above the mitral valve and close to the posterior border is a nodule 0.5 cm. in diameter, which is pale green in color. In the wall of the right auricle in the posterior side close to the interauricular septum is a similar nodule 1 cm. in diameter.

**Lungs:** Both are negative, except for edema and congestion. At the root of the left lung, presumably in mesenteric nodes, are two pale green tumor nodules about 1.5 cm. in diameter.

**Spleen:** Normal in color and consistency.

**Liver:** Pale, reddish-brown in color, normal in size and consistency. No tumor nodule found on close inspection.

**Pancreas:** There is a mass of large green glands at the head of the pancreas. On section these vary in color from a pale green to an olive green. Imbedded in the tail of the pancreas are two similarly colored nodules about 1 cm. in diameter.

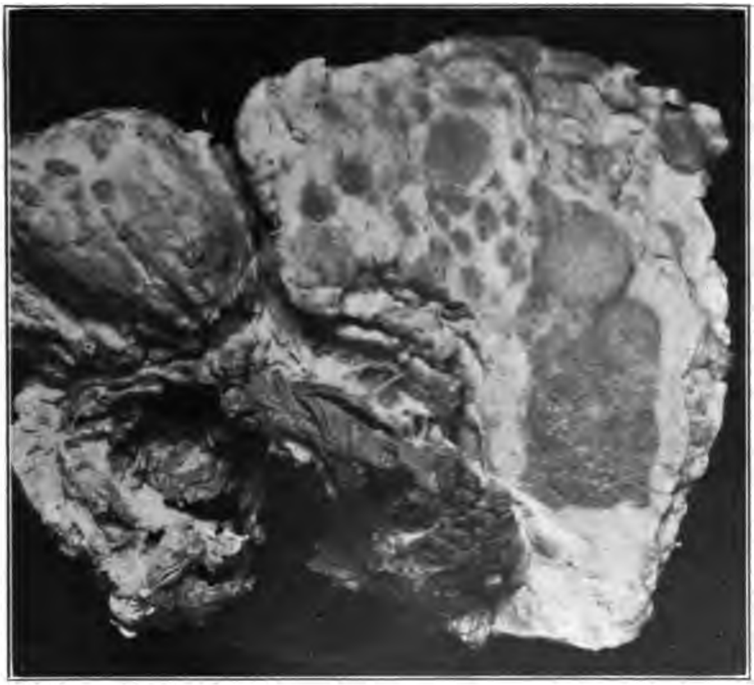

Fig. 2.—Breast and subjacent pectoral muscle showing tumor nodules of varying size and diffuse tumor growth (natural size).

Gastro-Intestinal Tract: Not opened.

Kidneys: Both kidneys are normal in size, pale in color, and each contain many nodules varying in size from 2 mm. to 1.5 cm. The largest are situated beneath the capsule and are flattened and soft in consistency. Smaller ones are distributed throughout from cortex to pyramids and are firmer in consistency. These nodules are of rather pale color but of a decided green tint. The larger ones show small red areas, presumably hemorrhages.

Adrenals: Both are normal.

Prevertebral lymph-nodes are markedly enlarged. On the left side, just above the bifurcation, of the aorta is a large mass of pea-green tumor tissue which is firmly attached to the periosteum covering the vertebræ. This mass is roughly hemispherical in shape with a base 5 cm. in diameter and a depth of 3 to 4 cm. Consistency is very firm.

Genitalia: Uterus large and has a partially contracted appearance of a recent delivery. Ovaries and tubes normal. Owing to lack of time, genitalia were not more closely inspected.

Fresh microscopic examination of the tumor shows it to be made up of small round-cells, a few of which contain many refractive granules. The fluid obtained by teasing and expressing glands contains many refractive globules which on exposure to fumes of osmic acid turn dark brown. Tissues preserved in Zenker's fluid, Fleming's fluid, formaldehyd solution, absolute alcohol, methyl-alcohol, acetone, Kaiserling and saturated solution of chloral hydrate.

Breasts: Both breasts and surrounding fat tissue are largely replaced by green tumor tissue. The growth consists of spherical and ovoid masses from a size just visible up to nodules 3 cm. in diameter. These nodules are of quite firm consistency. The color varies from a pale green to an olive green.

Anatomic Diagnosis: Chloroma with metastases to sternum, heart, bronchial nodes, kidney, pancreas and lymph-nodes.

The following pathologic report is made by Dr. Thomas Ordway, director of the Bender Laboratory:

## PATHOLOGIC REPORT

*Microscopic Examination.*—The tissue available for this examination[2] had been in part fixed in 10 per cent. dilution of liquor formaldehydi and in part preserved by the Kaiserling method, by Dr. S. B. Wolbach, who also, as indicated in the preceding protocol, carefully fixed tissue in Zenker's fluid, Flemming's fluid, absolute alcohol, methyl-alcohol, acetone, and saturated solution of chloral hydrate. This tissue will form the basis of a special pathologic study to be reported later by Dr. Wolbach, to whom I am indebted for the opportunity of this preliminary study of the formaldehyd and Kaiserling material. Large sections for topographical relations were imbedded in celloidin and stained by hematoxylin and eosin and by Van Giesen's connective tissue stain. Smaller fragments were imbedded in paraffin and stained by eosin and methylene-blue (Wolbach's colophonium modification) and hematoxylin and eosin. Teased preparations and frozen sections were examined unstained and stained by Scharlach R. and hematoxylin and Scharlach R.[3]

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[2] No bone-marrow or tissues, other than indicated below, were available for this study, owing to the limitations placed on the postmortem examination.

The new growth consists of nodules widely disseminated throughout the body, involving the breasts, sternum, bronchial and retroperitoneal lymph-nodes, the heart, pancreas, and kidneys (Figs. 2, 3 and 4). Most of the tumor masses appear as discrete circumscribed nodules. On microscopic examination, however, the tumor cells at the periphery, trail off into and diffusely invade the adjacent tissues. In some areas, even in the immediate neighborhood, the tumor growth is diffuse, with a tendency, however, to the formation of nodules.

Tumor-Character of Cells: The tumor consists of closely packed cells in general resembling those of the lymphocytic series; they are chiefly of the embryonic type. The nucleus is large, round or oval and the chromatin in many has a reticular appearance like that of the plasma cell; the nucleus also is in some eccentrically placed. The cyto-plasm is variable in amount, usually moderate, and takes a neutral or slightly acid or basic stain. Many of the nuclei are vesicular. There are also cells having fine and coarse granules with deeply staining reticular nuclei resembling the cells of the myeloma or myelocytes. Mitotic figures are frequent.

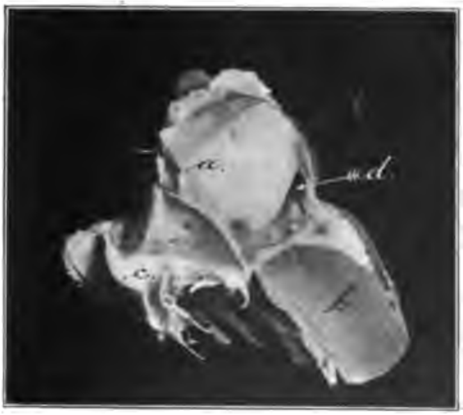

Fig. 3.—Heart: Fragment showing large tumor nodule in the wall of the auricle at *a*. This lies above and to the right of a portion of the mitral valve *c* and above the coronary vein *d;* part of the wall of the left ventricle is seen below, *b* (natural size).

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[3] I am indebted to Dr. Ellis Kellert for assistance in the sectioning, staining and photographic work. The photographs were made by Mr. J. A. Glenn.

Among the tumor cells is a delicate reticulum, similar to that of lymph-nodes, with endothelial cells and a variable amount of connective tissue. The latter depends in great part on the character of the tissue invaded. In parenchymatous organs little connective tissue appears in the tumor (Fig. 7). When dense fibrous connective tissue is invaded, it is split up and becomes incorporated in the tumor as stroma. The endothelial-like cells occasionally contain more than one nucleus, two to four, and resemble the type of giant-cell occurring in the scirrhous lymphoma or so-called Hodgkin's disease.

Eosinophilic cells are common, many resembling the eosinophilic myelocytes, and others the adult polymorphonuclear eosinophils. There are other cells in general resembling the tissue mast-cell so frequently seen in chronic inflammatory processes, and especially marked in tissues fixed in formaldehyd. The granules of many of these cells take a neutral or slightly acid stain; in some, however, the granules are large and appear like the so-called secretion granules in secretory epithelium. In others the granules are small and take a decidedly acid stain. There is no sharp distinction between these and the eosinophilic cells above mentioned.

Frozen sections and teased preparations of tumor nodules from the breast, preserved in Kaiserling solution, and still having a distinct apple-green color, unstained, show occasional cells with small refractive greenish granules. These, however, are not numerous; no evidence of them is found in frozen sections stained for fat by Scharlach R, while the normal fat of the breast and the degenerating gland cells take a brilliant scarlet color. It is possible that the examination of the fresh tissue and that immediately fixed in solutions containing osmic acid, may be of value in determining the origin of the pigment. Such a preliminary examination has already been mentioned by Dr. Wolbach in the above protocol. Sections fixed and stained in the usual way show no evidence of pigment.

Breast: The glandular tissue has in some areas been entirely replaced by the tumor cells, which appear in large and small masses resembling lymphoid tissue. Although the tumor cells tend to occur in circumscribed foci, from these "centers," the cells diffusely invade the adjacent fat, fibrous and glandular tissue. The glandular epithelium shows all stages of degeneration, the cells occurring in masses of varying size, separated and invaded by the tumor (Fig. 5). The clear, circular vacuole-like areas in the epithelial cells take on a brilliant red color similar to the adjacent fat tissue of the breast when frozen sections are stained by Scharlach R. Many of the ducts are dilated. In the vicinity of the degenerating glandular tissue, there is a varying number, in places an abundance, of large so-called compound granule-cells, endothelial-like cells containing droplets, and granules sharply stained red by Scharlach R.

**Large and small nerves are surrounded, apparently pressed on and invaded, by the tumor. Numerous ganglion cells, containing abundant yellow to brownish pigment, are similarly surrounded and show a varying degree of degeneration. Some are swollen and others are shrunken. The chromatic material is massed at the periphery of the cells. In many of the fibers, myelin sheaths have persisted, and in others there is marked degeneration. Among the necrotic glands and ganglion cells are varying numbers of polymorphonuclear leukocytes. In places the tumor cells are massed beneath the endothelium of the lymphatics. Where the tumor invades dense fibrous tis-**

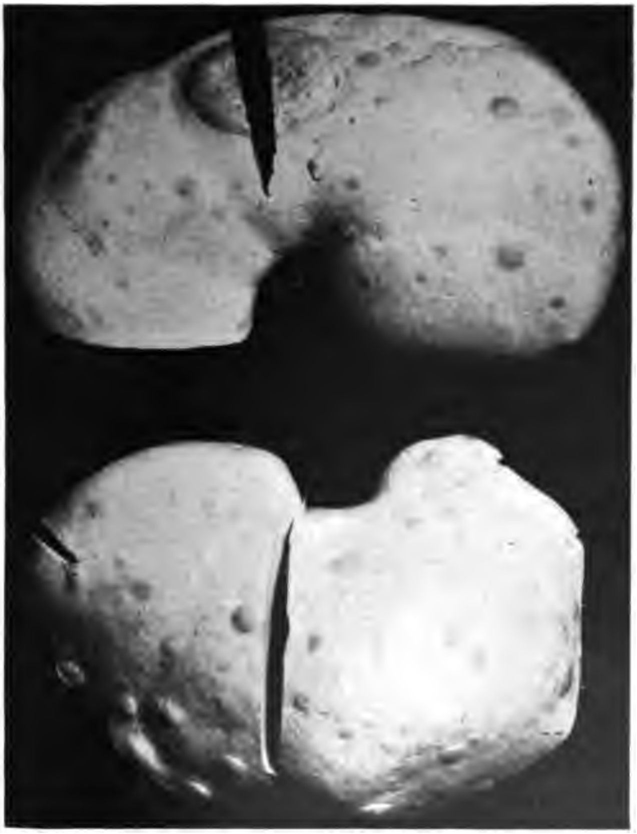

Fig. 4.—Kidney (outer surface): Scattered throughout and projecting from the cortex are numerous tumor nodules varying from 1/25 mm. in diameter up. The clefts indicate areas from which tissue has been excised for microscopical examination (natural size).

sue, bundles of collagen fibrils are split up and appear scattered throughout the tumor mass as stroma, the abundance of this depending on the variety of tissue invaded.

Sternum: The intercostal muscles, fascia and adjacent fat and fibrous tissue are diffusely invaded by the tumor, the cells of which infiltrate the perichondrium and rarely penetrate it (Fig. 6). In one place, cells resembling the tumor occur in close relation to the marrow of the sternal end of one of the ribs. The tumor-cells resemble those above described. A large lamellar, or Pacinian corpuscle, at the edge of the perichondrium, is found surrounded by tumor-cells. By the connective tissue stain the reticulum and stroma of the tumor in the neighborhood of the dense perichondrium, appear exceptionally abundant, and are clearly seen in the form of a delicate tracery among the tumor-cells. Most of this is due to the inclusion of the separated fibrillæ by the growth and invasion of the tumor. The muscle fibers invaded show a varying degree of degeneration. The wall of a large vein near a costal cartilage is involved by the tumor and a large number of tumor-cells are within the lumen (Fig. 6).

Lymph-Node: The normal structure of the lymph-node is obscured by the invasive tumor growth. Occasionally at one end of a node not completely invaded, cords of lymphoid tissue and lymph-follicles can be recognized. Here the sinuses are filled with large and small lymphoid cells and many eosinophils. These areas not involved by the tumor have the appearance of a marked, chronically inflamed lymph-node. Between these portions and those invaded by the tumor, the lymph-channels are distended by the tumor-cells. Here also an occasional blood-vessel is found, the wall infiltrated by tumor-cells which elevate the endothelium and are present mixed with red blood-corpuscles within the lumen.

Kidney: The tumor masses are in the form of large and small foci; the cells are not sharply circumscribed, but trail off among and diffusely infiltrate the glomeruli and tubules, which show all stages of degeneration (Fig. 7).

About and within the necrotic tubules are varying numbers of polymorphonuclear leukocytes; the cells of the convoluted tubules in the vicinity of the tumor are swollen, homogeneous, or vacuolated; in some are abundant hyaline droplets. Except for this excess of polymorphonuclear leukocytes, the tumor presents the general characteristics above described.

Heart: The tumor nodule in the wall of the auricle above the mitral valve (Fig. 3) has infiltrated the muscle-fibers, many of which are separated or replaced by the tumor; others show a varying degree of degeneration. The subepicardial fat and the wall of the large coronary vein are likewise invaded. The tumor-cells occur in masses beneath the endothelium of the vein, which in places is absent. In the subepicardial fat a large lymphatic is seen distended by tumor-cells.

Liver: Sections reveal no distinct tumor-nodules; in the periportal connective tissue, however, there are numerous cells of the lymphocytic series and eosinophils. In the sinusoids also, particularly near these areas, are similar cells in varying numbers. The hepatic cylinders contain a moderate to marked amount of yellowish granular pigment. The general appearance of the liver suggests rather that of myelogenous than of lymphatic leukemia.

The pancreas and adrenals are negative.

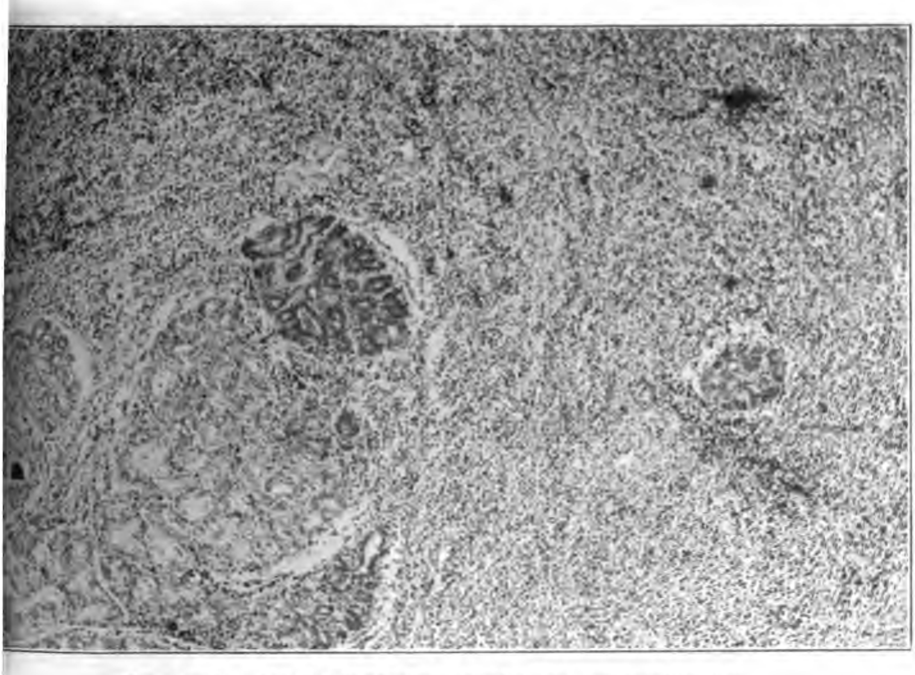

**Fig. 5.—Breast:** Glandular tissue invaded and separated by the tumor (x 65).

*Summary of Microscopic Findings.*—The tumor is widely disseminated and occurs in more or less circumscribed foci; it also shows diffuse and transitional forms of growth. The extension may be by way of the lymph or blood-streams as tumor-cells are found both beneath the endothelium and within lymphatics and blood-vessels.

The predominating cells are of the lymphocytic series and occur in a delicate reticulum; this contains a varying amount of connective tissue, depending on the nature of the tissue invaded. Cells of the myelocytic series and endothelial cells, single and multinucleated, occur in varying numbers.

Tissues invaded by the tumor undergo different forms of degeneration with resulting acute and chronic inflammatory reaction of slight to moderate degree. This is characterized by the presence of polymorphonuclear leukocytes, eosinophils, mast-cells, lymphoid and endothelial cells; many of the latter are phagocytic for products of cell-degeneration.

The nature and source of the pigment is not evident, but preliminary observations show that this may be determined by subsequent study of fresh or suitably fixed tissue. Such pigment is not found in tissue fixed and stained in the usual manner.

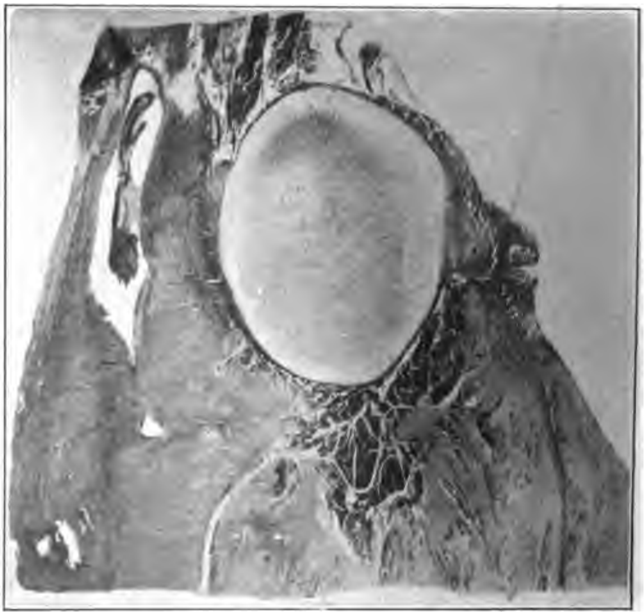

Fig. 6.—Sternum: Section showing costal cartilage almost surrounded by tumor, the cells of which have invaded the intercostal muscle, adjacent fat and fibrous tissue and the wall of a large blood-vessel (to the left), and are present in large numbers in the lumen (x 5).

Pathologically the disease is characterized by growths, most frequently multiple, in connection with the bones of the face, orbit and other regions. Macroscopically the growths vary in color from a dark olive to a very light pea green which fades rapidly on exposure to air or the various fixation fluids. The color is probably due to a lipochrome which is found in extremely variable amount even in the cells of the primary growth.

The blood-picture is held by Dock, Warthin, and Jendrassik, among others, to be characteristic, the lymphoblasts largely predominating, with a moderate to pronounced anemia, usually with extremely few megaloblasts. The neutrophilic myelocytes in the majority of cases are relatively in small numbers. Toward the latter stages of the disease degenerated leukocytic forms, together with erythrocytic deformity and polychromia without stippling, make their appearance.

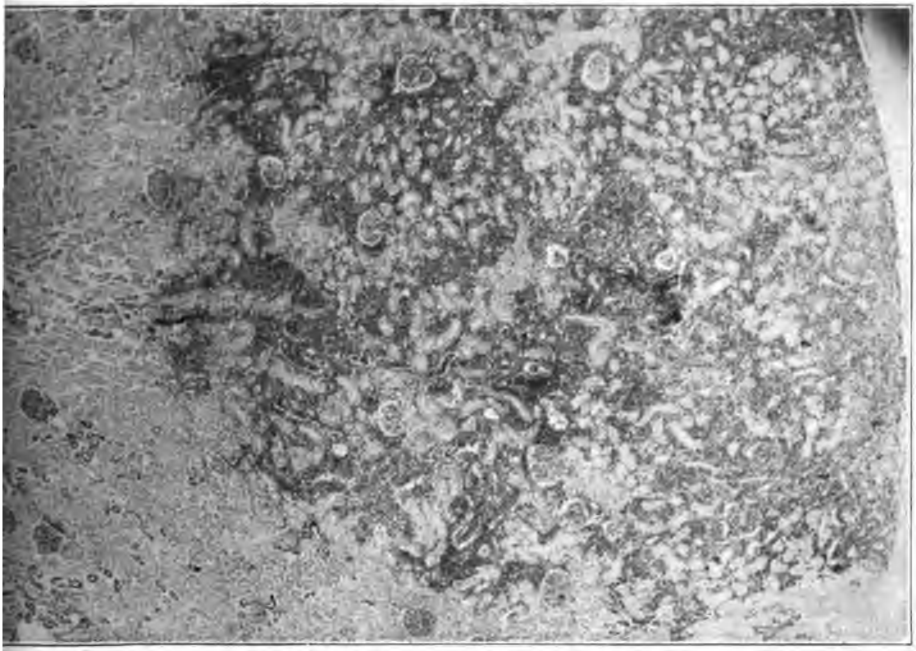

Fig. 7.—Kidney: Tumor diffusely infiltrating cortex (x 35).

The ocular manifestations are many and varied, and, therefore, will not be considered in detail, as the cases here reported are characteristic.

I wish to express my thanks to Dr. Ordway for his report and the many kindnesses shown me, to my residents Drs. McSorley and Munson for their interest, and to Dr. Lochner for the opportunity of recording this case.

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# MEMORANDA

the turnstile of astigmatism." This fact is so generally recognized as not to require confirmation at this time, yet many text-books make no mention of it. The "hypermetropia acquisita" of Donders is the only supposed change in the refraction of the eye of which note is generally taken. MacNamara, quoted by Randall, urges that young hyperopes, even those with absolute hyperopia, should not be given correcting glasses or even under-correcting lenses for constant wear, asserting that the normal development of the eye, whereby the hyperopia would be outgrown, might thus be interfered with, and the defect rendered permanent. Randall, having had the same thought, and having acted on it, did not observe a decrease in the hyperopia in a single instance. Nettleship states that hyperopia always dates from birth and does not afterward increase, except slightly in old age; but it may diminish and give place to myopia by elongation of the eye.

Jackson sees in the occurrence of astigmatism in 41.8 per cent. of myopes and only 17.2 per cent. of hyperopes, a connection between astigmatism and myopia. He further states that there is little reason to doubt the correctness of the common belief, that the majority of cases of astigmatism remain the same throughout life. He records eighteen cases of progressive hyperopic astigmatism. In all, there was an increase in the degree of the hyperopic astigmatism. He is of the opinion that the frequency of these cases would be about 2 per cent. of all cases seeking relief on account of anomalies of refraction. The cases, in which after the lapse of an equal length of time, the use of a mydriatic has shown no change in the amount of the astigmatism outnumbered them very decidedly, and cases of mixed and myopic astigmatism are relatively, though not absolutely, more frequently progressive. He has encountered no case of regressive astigmatism—none in which the astigmatism had diminished so much as ½ D., though it is not improbable that such cases occur. In most of the cases there was an increase in the hyperopia in all of the meridians. In six of the cases there was actual diminution of the hyperopia in one of the principal meridians of one or both eyes. He has not found that the liability to change in the amount of astigmatism is confined to any one particular period of life, though such changes may be more frequent in early life.

Emerson also reported a case of progressive hyperopic astigmatism.

Theobald has recorded a case in which there occurred an increase of hyperopia with an increase of astigmatism, a case of increase of myopia with an increase of astigmatism, and a case of myopic astigmatism becoming mixed astigmatism. He believes such cases to be probably of infrequent occurrence. Randall doubts the occurrence of hyperopia acquisita of advanced life in the sense referred to by Donder.

According to Thorington, astigmatism may develop from any disease or injury that will cause a lengthening. shortening, or inequality in one or more of the meridians of the cornea or lens. The same author refers to the development of compound myopic astigmatism from low degrees of compound hyperopic astigmatism and in this connection states that it is well known that hyperopic astigmatism is a congenital defect.

Alger refers to the development of myopia from hyperopia and also the acquired hyperopia of old age.

Duane is the only author whose writings contain any extended reference to the phase of refraction with which this paper has in particular to do. Regarding astigmatism, he says: "Once developed, it remains, on the whole, practically stationary during childhood, youth and early manhood. Changes do take place in it from time to time, but they are inconstant and usually slight, and there is no special rule governing the way in which they develop. After middle life, however, there is a tendency for astigmatism to change its character. Astigmatism with the rule tends to become less, or is sometimes converted into astigmatism against the rule, and astigmatism against the rule often develops where no astigmatism existed before, so that in old age astigmatism against the rule is the most common variety."

An analysis of the changes of refraction occurring in 550 eyes was made. The eyes were divided into three groups representing age periods at which changes are most likely to occur, namely: patients under 20, between 20 and 45, and over 45. As the study progressed, further subgroups seemed desirable and were made. One hundred and fifty-five patients (28 per cent.) came under the first group, 322 (55 per cent.) under the second group and ninety-two (17 per cent.) under the third group. In 222 over ten years (in many instances more than fifteen years) had elapsed between the first and last refraction tests, in 320 between five and ten years had elapsed. The cases were all from private records. With few exceptions in persons under 50 years of age all the tests were made under cycloplegia, usually secured by the use of atropin; in between fifty and fifty-five either no cycloplegic or homatropin was used. In most cases the glasses had been worn constantly.

No eyes were included which showed incipient cataract, glaucoma, present or past evidences of inflammation either in the anterior or posterior segment of the globe or in which a developing pterygium or a chalazion was present. This was done because it was not the purpose to show the effects of inflammation or degenerative processes on the refraction of the eye.

The class of patients were those usually met in private practice.

The cases were analyzed to show: no change in HaH,[1] no change in MaM, no change in HAs; no change in MAs, no change in M or H; increase in static refraction M and H; increase in the static refraction with increase or decrease in the astigmatism; decrease in the static refraction with an increase or decrease in the astigmatism; MAs+HAs analyzed to show changes occurring in the principal meridians; low-grade ametropia going over into MAs+HAs, and all possible transitional forms; development of astigmatism where no astigmatism had before existed, loss of total astigmatism.

In order to avoid confusion or misunderstanding, in the summary the changes in the static refraction and in the astigmatism in compound astigmatic eyes have been classified according to the nature of the ametropia.

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[1] Abbreviations: M=myopia; H=hyperopia; HAs=simple hyperopic astigmatism; MAs=simple myopic astigmatism; HaH=compound hyperopic astigmatism; MaM=compound myopic astigmatism; MAs+HAs=mixed astigmatism.

The accompanying table summarizes the analysis:

| | Number of Cases. | Percentage of All M or H Eyes. | Percentage of H or M Eyes Which Underwent a Change. | Percentage of HaH or MaM Eyes Which Underwent a Change. | Percentage Under 20 Years of Age. | Percentage from 20 to 45 Years of Age. | Percentage Over 45 Years of Age. |
|---|---|---|---|---|---|---|---|
| Increase of H with increase of As | 36 | 9 | 12 | 16 | 17 | 53 | 30 |
| Increase of H with decrease of As | 44 | 11 | 15 | 19 | 18 | 60 | 22 |
| Decrease of H with increase of As | 116 | 30 | 40 | 51 | 36 | 57 | 7 |
| Decrease of H with decrease of As | 32 | 8 | 11 | 14 | 19 | 57 | 25 |
| Increase of M with increase of As | 16 | 24 | 31 | 44 | 37 | 56 | 6 |
| Increase of M with decrease of As | 9 | 14 | 17 | 26 | 22 | 67 | 11 |
| Decrease of M with increase of As | 8 | 12 | 15 | 23 | 37 | 63 | 0 |
| Decrease of M with decrease of As | 2 | 3 | 4 | 6 | 0 | 100 | 0 |
| Increase of H | 40 | 10 | 14 | .. | 22 | 38 | 40 |
| Decrease of H | 24 | 6 | 8 | .. | 30 | 45 | 25 |
| Increase of M | 16 | 24 | 31 | .. | 7 | 93 | 0 |
| Decrease of M | 1 | 2 | 2 | .. | 0 | 0 | 100 |
| HaH no change | 51 | 13 | .. | .. | 12 | 78 | 9 |
| MaM no change | 11 | 17 | .. | .. | 18 | 27 | 54 |
| H no change | 46 | 12 | .. | .. | 26 | 54 | 20 |
| M no change | 3 | 5 | .. | .. | 0 | 100 | 0 |
| HAs or low HaH or MaM becoming MAs+HAs | 22 | .. | .. | .. | .. | .. | .. |
| Various other transitions | 64 | .. | .. | .. | .. | .. | .. |
| HaH with change in only one element | 32 | .. | .. | .. | .. | .. | .. |
| MAs+HAs with change in M or H meridian | 28 | .. | .. | .. | .. | .. | .. |
| Development of As in H | 30 | .. | .. | .. | .. | .. | .. |
| Development of As in M | 1 | .. | .. | .. | .. | .. | .. |
| Loss of total As | 9 | .. | .. | .. | .. | .. | .. |

Summary: Total H eyes, 389; total HaH eyes, 279; total M eyes, 46; total MaM eyes, 66. Eighty per cent. of the eyes tabulated underwent a change either in the static refraction or in the amount of astigmatism.

### CHANGES IN COMPOUND ASTIGMATIC EYES

The largest percentage of change in HaH occurred in the group in which there is a decrease in the H with an increase of As. This took place in 51 per cent. of all HaH showing a change in the amount of H or As; and in 30 per cent. of all hyperopic eyes tabulated. The second largest percentage of change occurred in HaH in the group in which there is an increase in the H with a decrease in the As. This took place in 19 per cent. of all HaH eyes showing a change in the amount of H or As; and in 11 per cent. of all hyperopic eyes tabulated. In MaM, the largest percentage of change occurred in the group in which there is an increase in the M with an increase in the As. This took place in 44 per cent. of all

MaM eyes showing a change in the amount of M or As; and in 24 per cent. of all myopic eyes tabulated. The second largest percentage of change in MaM eyes occurred in the group in which there is an increase in the M, with a decrease of the As. This took place in 26 per cent. of all MaM eyes showing a change in the amount of M or As.

Considering these groups in the sense of a change in the static refraction it will be seen that in eyes with a compound astigmatic ametropia, there was an increase in the static refraction with an increase in the astigmatism in 48 per cent.

### AGE PERIOD AT WHICH CHANGE OCCURRED

Fifty-seven per cent. of the changes in HaH occurred between the age period of from 20 to 45 years; 23 per cent. under 20 years, and 20 per cent. over 45 years. In MaM 71 per cent. occurred between 20 and 45, 24 per cent. under 20, and 5 per cent. over 45. While these figures show that far the greatest number of changes occurred in the age period of from 20 to 45 years, they are probably of no significance, as the same preponderance of numbers is seen in this same period in the cases of HaH which underwent no change. They show, however, that changes in M are of infrequent occurrence after the forty-fifth year, and that they occur in H with much greater frequency, but still with much less frequency than before the forty-fifth year.

Of the total number (279) of HaH, 228, or 80 per cent., underwent a change in the relation between the amount of H and the amount of the As; and of the forty-six MaM eyes thirty-five, or 76 per-cent., underwent a similar change. This would indicate that there is no greater tendency for MaM to change than there is for HaH to change. Of the twenty-eight cases of MAs+HAs, twenty-five, or 89 per cent., underwent a change in the relation between the amount of H and M in the principal meridians. Of the thirty-two cases of HAs twenty-nine, or 90 per cent., underwent a change in the refraction of the principal meridians. Of the ten cases of MAs ten, or 100 per cent., underwent a change in the principal meridians.

This shows conclusively that simple and mixed astigmatism are the least stable of the ametropic refractive states. In MAs+HAs the eyes in which there was an increase in the M with a decrease in the H; an increase of the H with a decrease of the M; an increase of the M with an increase of H, and an increase of M with no change in H, were almost evenly divided. Twenty-two eyes with a low compound As or a simple As went over into mixed As and *vice versa,* nine eyes with MAs+HAs became MaM, HaH, MAs.

### CHANGES IN HYPEROPIA

Of 126 hyperopic eyes, in patients over 45 years of age in which the average age at which the final test was made was 53 years, in fifty-one, or 40 per cent., there was a decrease in the H, in forty-one, or 33 per cent., there was an increase in the H, and in thirty-one, or 27 per cent., there was no change. Because of these variations from the generally accepted views a second series of fifty-five eyes, in which the average age at which the first test was made was 55 years and the final test 63 years, was analyzed. In 45 per cent. there was an increase in the H, in 27 per cent. there was a decrease, and in 27 per cent. there was no change. Here, again, we see that the eyes in which no increase occurred overbalance those in which an increase occurred. While these figures are insufficient in number to warrant a dogmatic statement there does not seem to be any greater tendency at these age periods for the H to increase than for it either to remain stationary or decrease.

The average amount of increase of H equaled ⅜ D. The average amount of decrease in H equaled ⅝ D. In the group showing an increase in H, one eye in which a rather rapid increase of 3 D. occurred was omitted, and in the group in which a decrease occurred two eyes, in one of which the change equaled 2 D. and the other 6 D., both in the same individual, were omitted, as the change occurred rather rapidly and it was considered probable that it was due to a pathologic cause. If they be included then the average increase was ½ D., and the average decrease 1 D.

Including in the group "H no change" cases of HaH in which no change whatever took place, and cases of H in which no change occurred, there were ninety-seven cases, or 25 per cent. of all of the hyperopic eyes tabulated. Of the pure cases of H there were twenty-four, of these four, or 16 per cent., underwent no change, twelve, or 50 per cent., showed an increase, and eight, or 33 per cent., underwent a decrease.

### CHANGES IN MYOPIA

Included in the group "M no change" are the same class eyes as were included under the same heading for H. Of these there were fourteen. Of the pure cases of M there were four. All of the latter underwent an increase.

These figures indicate that H is more stable than M, and that whereas in M there is invariably an increase in H there may be either an increase or a decrease.

### ACQUIRED ASTIGMATISM

Astigmatism was acquired in thirty-one eyes. In all but one instance, the eyes acquiring the astigmatism were hyperopic, or 8 per cent. of the hyperopic eyes. Sixteen, or 51 per cent., of the individuals were under 45 years of age. The average age in this group at which the astigmatism was first present was 32 years. In seven, or 44 per cent., the axis of the astigmatism was against the rule; in seven, or 44 per cent., it was with the rule; and in two midway between 90° and 180°. The average amount of astigmatism equaled a little less than ½ D. In fifteen, or 49 per cent., the individuals were 45 years of age or over. In this group the average age at the time the astigmatism was discovered was 53 years. All the individuals were hyperopic. In thirteen, or 87 per cent., the axis was against the rule. The average amount of As equaled a little less than ½ D. These figures indicate that astigmatism may develop at any decade and occurs with equal frequency in the third as in the fifth decade, and that after the forty-fifth year; in the majority of cases the axis was against the rule, while before this period the axis was as often with as against the rule.

### LOSS OF ASTIGMATISM

The total astigmatism was lost in nine eyes; in four, or 44 per cent., in individuals under 45 years of age. In this group, the average age at which the loss was determined was 36 years. All the eyes were hyperopic. In three, or 75 per cent., the astigmatism had been with the rule. In five, or 56 per cent., the individuals were over 45 years of age. In this group, the average age at which the loss of astigmatism was discovered was 53 years. All were hyperopic. In three, or 60 per cent., the axis was with the rule, and in two, or 40 per cent., it was against the rule. The average amount of the astigmatism lost was ½ D.

These figures indicate that the total astigmatism probably may be lost in any decade, but that this occurs most frequently in the later decades and possibly represents a transitional stage to astigmatism against the rule, as in 60 per cent. the axis of the astigmatism which had existed was with the rule.

## CHANGES IN MIXED ASTIGMATISM

Of twenty-eight cases of mixed astigmatism, twenty-five, or 89 per cent., underwent a change in one or both elements, and in three, or 11 per cent., no change occurred. There was an increase of M with a decrease of H in six eyes; an increase of H with a decrease of M in seven eyes; an increase in both M and H in six cases; and an increase of M with no change in H in six. The tendency is for both meridians to change, but the most frequent change is for the myopic meridian to increase; but the most frequently associated change was an increase in the hyperopic meridian associated with a decrease in the myopic meridian.

## CONCLUSIONS

1. Ametropia tends to change in degree even when the error is carefully and repeatedly corrected and the glasses worn constantly.
2. Almost every conceivable change may occur.
3. The tendency is in the direction of an increase in the static refraction with an increase in the astigmatism.
4. In compound hyperopic astigmatism the most frequent change is a decrease of the hyperopia with an increase in the astigmatism.
5. In compound myopic astigmatism the most frequent change is an increase in the myopia with an increase in the astigmatism.
6. There is no greater tendency for compound myopic astigmatism to change than there is for compound hyperopic astigmatism to change.
7. Simple myopia (rare) is never a stable condition, it almost invariably increases in amount.

8. Simple myopic astigmatism is the least, and simple hyperopic astigmatism the next least stable of astigmatic errors.

9. Simple hyperopia sometimes remains constant, but is subject to undergo both an increase and decrease.

10. In compound astigmatism the static refraction may change without a change taking place in the astigmatism and *vice versa.*

11. In mixed astigmatism, the myopic meridian most frequently changes and shows an increase, although an increase of the hyperopic meridian with a decrease in the myopic meridian in the same eye probably occurs with the most frequency.

12. Astigmatism is often acquired in hyperopic eyes, and as frequently in the third as in the fifth decade. Before the forty-fifth year the axis of the acquired astigmatism is as frequently with as against the rule, but after the forty-fifth year it is against the rule in 85 per cent. of patients.

13. The total astigmatism may be lost probably in any decade, but more commonly in the later decades and at this time probably represents a transitional stage to an astigmatism against the rule.

14. A change in the axis of astigmatism occurs in about 50 per cent. of the cases.

15. The changes in the refraction occur with most frequency in the age period of from 20 to 45 years.

16. After the forty-fifth year changes in the amount of hyperopia in compound hyperopic astigmatism occur much more frequently than do similar changes in compound myopic astigmatism.

17. There is no greater tendency toward an increase in hyperopia between the fifty-fifth and sixty-fifth years than between the forty-fifth and fifty-fifth years.

1819 Spruce Street.

## MEMORANDA

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# REFRACTION CHANGES IN DIABETES *

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Since Horner first called attention to the subject and reported two cases to the Ophthalmologic Society of Zurich in 1873, a number of examples of sudden change in the refraction of glycosuric patients have appeared in the literature. The text-books have had little to say on the subject and what is to be found in the standard works is misleading.

The object of this paper is to call attention once more to the fact that the refraction not only may be increased in diabetes, but that it may be diminished; that both changes may occur in the same patient; and that the changes bear no necessary relation to the amount of sugar in the urine. As is well known, diabetes sometimes causes a true paralysis of accommodation, and in a number of cases of refraction change due to this disease there has been either a limited range, or a more or less definite paralysis. One author, at least, it seems to us, has confused a case of increased hyperopia with paralysis of accommodation. This case will be referred to later. Cases of increase in the refraction of the eye (increased myopia or diminished hyperopia) in glycosuria have been reported by Grimsdale, Appenzeller, Hirschberg, de Schweinitz, Neuberger, Groenouw, and others; but with these we have no present concern. The following cases from the literature, it seems to us, offer trustworthy evidence of diminished refraction due to diabetes:

HORNER'S[1] CASE.—The patient was a woman, aged 55, who since August, 1870, had suffered with diabetes mellitus, passing an enormous quantity of urine, heavily loaded with sugar.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

[1] Horner: Klin. Monatsbl. f. Augenh., 1873, xi, 488.

She had been wearing no glasses, except for near work at night, when she used R. E. +0.75 Sph. L. E. +1.50 Sph. By the end of October her sight was failing and her glasses no longer were suitable. On November 19, she accepted, both eyes, +2.75 Sph. R. V. =5/5, L. V. =6/5; +1.50 Sph. was added for near. There was no intra-ocular lesion. As the diabetes improved the convex lenses no longer suited. Jan. 13, 1871, she accepted, both eyes, +0.75 Sph. Vision was the same as above noted, and the correction for presbyopia also the same (+1.50).

RISLEY'S[2] CASE 1.—The patient was a woman, aged 49, who had improved under treatment, and developed dimness of vision only after her symptoms of diabetes were largely relieved. Her old lenses (+2.75 Sph.) no longer served, and her vision was reduced to 1/5 in the right eye, and 1/4 of normal in the left eye; and there was no range of accommodation. With mydriatic, the following refraction was revealed:

R. E. +5.00 Sph. +1.25 Cyl. Ax. 75°, V. =6/6, L. E. +6.00 Sph. +0.75 Cyl. Ax. 105°, V. =6/6.

Ten days later her vision was again dim with glasses, and she accepted:

R. E. +3.00 Sph. +1.25 Cyl. Ax. 75°, V. =6/6, L. E. +3.50 Sph. +0.75 Cyl. Ax. 105°, V. =6/6, and accommodation was still absent.

Fifteen days later she accepted:

R. E. +1.25 Sph. +1.25 Cyl. Ax. 75°, V. =6/6, L. E. +1.50 Sph. +0.75 Cyl. Ax. 105°, V. =6/6.

Through several months of careful observation no further change was noted, though the patient steadily grew worse, with persistent glycosuria.

RISLEY'S[2] CASE 2.—The patient, a man of 74, had suffered with diabetes for at least six years, when he complained of dimness of vision. His refraction proved to be:

R. E. +1.50 Sph. +1.25 Cyl. Ax. 180°, V. =6/7.5, L. E. +1.25 Sph. +1.25 Cyl. Ax. 180°, V. =6/10.

From this time (August, 1896) until April, 1897, there was no change in the eyes. April 23, he complained of dimness of vision for distance and inability to read even large type with his reading lenses. His diabetes at this time was improved. He now accepted:

R. E. +2.50 Sph. +1.25 Cyl. Ax. 180°, V. =6/7.5, L. E. +2.50 Sph. +1.25 Cyl. Ax. 180°, V. =6/10.

May 20, he returned complaining that his vision was blurred with the distance lenses after a few days' use, and that he must hold his book too close in reading. He accepted his old glasses and had vision: R. E. 6/7.5?, L. E. 6/10.

CARPENTER'S[3] CASE.—The patient, a woman aged 51, had consulted Carpenter six years before on account of presbyopia.

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[2] Risley: Tr. Am. Ophth. Soc., 1897, p. 121.
[3] Carpenter: Abstracted in Ophth. Rec., June, 1902.

She now complained of sudden inability to see clearly at a distance or to read with her near glasses. Her refraction, which had been a slight myopic astigmatism, was now hyperopic: R. E. +1.50 Sph. V. = 6/6, L. E. +2.50 Sph. V. =6/6. With +2.25 Sph. added she read Jaeger 1 at 30 cm. The urine had contained a large amount of sugar four weeks previously; and a reduction in the amount of sugar, under strict dieting, was coincident with the failing vision. There was no lesion of media or fundi. In three weeks, vision again blurred with glasses and she showed a myopia of 0.50 D.

GALLUS'[4] CASE.—The patient was a man of 51, who had had symptoms of diabetes for some time but had been under treatment only two weeks. He had been seeing well at a distance and wearing +2.00 spheres for reading. Recently his distant vision had failed and he saw better for distance with his reading lenses which no longer served their former purpose. His refraction, on March 28, 1905, was found to be:

R. E. +1.50 Sph. +0.50 Cyl. Ax. 180°, V. =5/8, L. E. +2.00 Sph. +0.50 Cyl. Ax. 180°, V. =5/5. +2.00 Sph. added enabled him to read the finest print. The eyes were in every way normal except for a slight pallor of the temporal halves of the disks and slight narrowing of the form fields with overlapping of red and green. May 23, the sugar was absent, and the patient was using his old correction (+2.00 Sph.) for reading. Refraction now was:

R. E. +0.50 Sph. +0.50 Cyl. Ax. 180°, V. =5/7, L. E. +0.50 Cyl. Ax. 180°, V. =5/5. With +2.00 Sph. added for near he was able to read the finest print. The fields were now noted as being wider but not normal.

LICHTENSTEIN'S[5] CASE.—This is of especial interest, inasmuch as it exhibits diabetes in a youth of 17 years, with complete paralysis of accommodation and an increase in hyperopia, proved by repeated examinations under cycloplegia. This patient showed, on March 8, 1906, a slight anisocoria, and reduced vision. His eyes were otherwise normal. His manifest refraction was +1.50 Sph., both eyes, V. =6/6. This agreed with retinoscopy. A +12.00 Sph., both eyes, was required to read Jaeger 1 at 9 cm. The diagnosis of complete double-sided paralysis of accommodation was made, for which no cause could be discovered by the ophthalmologist or neurologist, except diabetes, which was established by the internist. There was 4.5 per cent. of sugar in the urine, of which six liters were passed daily. The diabetes increased in spite of treatment. On the following day the manifest refraction was +2.50 Sph., and +13.00 Sph. was required for near. Homatropin was then used, with the same finding; namely, +2.50 Sph. Two days later, under atropin cycloplegia, hyperopia =3.50. There was no change in the next two weeks, during which time the sugar was reduced and the patient gained in weight. In another week the refraction was still +3.50 Sph., and now a +11.00 Sph. was sufficient, instead of +13.00, to bring the near point to 9 cm. Thereafter a steady strengthening of the accommodation was manifested by the progressively smaller convex lenses required, until two months later, when a +5.00 Sph. sufficed. During this time the hyperopia was found to be 3.00, 2.50, 2.00, and finally 1.50, the latter estimate being with atropin cycloplegia. The thoroughness of the use of cycloplegia in this case is attested by the patient's refusing to return for further examination on account of his fear of atropin.

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[4] Gallus: Ztschr. f. Augenh., 1906, xv, 319.

[5] Lichtenstein: Ztschr. f. Augenh., 1906, xvi, 330.

**Landolt's[6] Case.**—No mention is made of the use of a cycloplegic, and the amount of hyperopia developed is too small to be conclusive. The patient was a woman, the subject of a traumatic diabetes, who had arrived at an age when the accommodation is almost *nil*, and in whom the refraction was made repeatedly with great care, according to the authors' statement. A hyperopia of 0.50 developed, and the refraction showed variations in direct proportion to the amount of sugar in the urine, finally returning to emmetropia.

**Doyne's Case.**—Unfortunately we do not know the refraction of Doyne's[7] patient, a physician aged 40, previous to his acute diabetes; but the subsequent history points strongly to an increase of hyperopia, later approximating emmetropia with the cure of the diabetes. Early in the course of the disease, when vision had become very dim, hyperopia was 3.00 D. With improvement in the patient's general condition the error gradually lessened until, when the urine was entirely free from sugar, the refraction was +0.75 Sph. with homatropin cycloplegia.

**Jackson's[8] Case.**—A woman, aged 52, came with a history of diabetes of some years' standing. Recently the amount of sugar had greatly increased and the general condition was worse. Until a few weeks before, the patient's distant vision had been good. At the time of her visit vision was 4/60. With R. and L. +2.25 Sph., vision was 4/4. These lenses were prescribed for distance, and +2.00 added for near. Three months later she returned complaining that she could not see with the distance glasses and the reading lenses were unsatisfactory. Vision without lenses was now 4/4, and the same with +0.50 Sph. She was given +2.75 Sph. for near work and there was no change six weeks later, but she grew rapidly worse and soon died.

**Lundsgaard's[9] Case 1.**—The patient was a woman of 57, who was given +0.50 Sph., both eyes, V. =6/6, in March,

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[6] Landolt and De Wecker: Traité complet d'ophthalmologie, iii, 388.

[7] Groenouw: Augenleiden bei Diabetes Mellitus, 1907, p. 46.

[8] Jackson: Tr. Acad. Ophth. and Oto-Laryngol., 1905, p. 221.

[9] Lundsgaard: Ztschr. f. Augenh., 1907, xvii, 156.

1902. No lesion was found in her eyes at this time. In July, 1905, she developed abnormal thirst and hunger and passed three to four liters of urine daily. Numerous examinations of the urine during 1905 revealed no sugar. A trace was present in February, 1906. In August, 1906, there was 7.75 per cent. of sugar. With restricted diet the percentage fell to 2.25 per cent. in ten days. August 24, the patient noted a dimness of vision which remained unchanged for eight days. Examination revealed R. E. +2.50 Sph. V. =6/6; L. E. +2.00 Sph. V. =6/6. No lesion of either eye. Accommodation was recorded as corresponding to the patient's age. The sugar diminished in quantity during September, and on September 15 the refraction was: R. E. +2.00 Sph., L. E. +1.50 Sph. By October 14 sugar was absent, and on November 19 the cloudy vision had disappeared. She read with her old reading lenses, and the refraction was the same as in 1902 (hyperopia =0.50), with normal vision.

LUNDSGAARD'S[10] CASE 2.—The patient was a woman aged 50. She had had diabetes for half a year. July 8, 1907, she suddenly found that her glasses (+1.50 Sph.) caused blurring. Her refraction was now found to be R. E. +3.00 Sph. V. =6/6, L. E. +3.50 Sph. V. =6/6. Accommodation was normal and ophthalmoscopic findings normal. Tension normal. Two days later the dimness of vision vanished and she saw with her old glasses. On July 15, the refraction was recorded as emmetropic. V. =6/6. The sugar, which was 6.2 per cent. on June 26, fell to 2.5 per cent. on June 29, 1.7 per cent. on July 2, 0.3 per cent. on July 5, 0.2 per cent. on July 8; thus we note a sudden increase in hyperopia occurring some days after the sugar had fallen from a high percentage to a mere trace. The accommodation remained normal throughout. The author states that he had no opportunity to use atropin.

LUNDSGAARD'S[10] CASE 3.—This is unique in the thoroughness with which it has been studied and reported. A woman, aged 50, stated that her vision had been good until presbyopic symptoms appeared one year previously. She had been given a glass for near work which was later increased to +1.50 Sph. For a half year she had suffered with diabetes, and the sugar had increased from 2.5 per cent. to 6 per cent. At the time of her first visit (Nov. 5, 1909) she was disturbed by blurring of distant vision and inability to use her reading lenses. She had headache and saw objects distorted, curved and angular, with the left eye. Refraction showed R. E. +1.50 Sph. V. =6/6, L. E. +2.00 Sph. V. =6/12. With +3.50 Sph. both eyes, she could read Jaeger 1. With the left eye the letters were out of line, some higher, some lower. The Schiötz tonometer revealed normal tension and the Javal ophthalmometer showed corneal astigmatism. The right pupil was slightly smaller than the left. Both reacted normally. The media were clear except for a mass of grayish, punctate flecks, partly vacuoles, in the right eye. They appeared to be in the front part of the lens, distinctly seen with the corneal loupe. The left eye showed no such appearance when the pupil was widely dilated with homatropin.

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[10] Lundsgaard: Klin. Monatsbl. f. Augenh., 1910, xlviii, 38.

November 15, R. E. +2.50 Sph. V. =6/6—; L. E. +2.50 Sph. V. =6/12.

November 18, sugar 0.85 per cent.

November 22, no change; atropin, left eye.

November 30, R. E. +1.00 to +1.50 Sph. V. =6/6—; L. E. +2.00 Sph. V. =6/12.

November 15, R. E. +2.50 Sph. V. =6/6—; L. E. +2.50 Sph. V. =6/12—.

December 30, refraction and vision were unchanged. Flecks and vacuoles unchanged. Sugar 2.3 per cent.

January 16, 1910, no change in refraction or vision. Atropin was used each time in the left eye in order to study the lens changes with a dilated pupil. The opacities seemed less numerous than formerly and very superficial and mostly in the periphery.

February 1, the right eye was reported emmetropic with vision 6/9; the left eye (manifest and under atropin) +0.50 Sph. V. =6/12. The distortion of objects in the left eye was unchanged, though the usual tests with straight lines for metamorphopsia showed no abnormality. During these numerous examinations, the ophthalmometer and tonometer readings showed at no time any change in tension or corneal astigmatism. The patient said that she saw as well as before the development of her diabetes, though the lens opacities remained unchanged.

KNAPP'S[11] CASE 1.—The patient was a woman of 47, who was found to be emmetropic by skiascopy, and accepted no lens for distance, in December, 1907. She was given +1.25 Sph. for her presbyopia. Oct. 26, 1908, she reported that she had suffered with diabetes since the first of the year, and for several days her vision had been poor for distant and near objects. Her vision was 6/36. Skiascopy showed hyperopia of 2.00 D., and +2.00 Sph. gave 6/9 vision. With +4.00 Sph. she read finest type at 30 cm. Ophthalmoscopy revealed red disks, but no lesion. November 9, there was no sugar in the urine and she accepted: R. E. +1.00 Sph. V. =6/6; L. E. +0.50 Sph. V. =6/6; agreeing with skiascopy.

November 23, R. E. +0.50 Sph.; L. E. +0.25 Sph. No sugar.

February 13, 1909, R. E. +0.50 Sph. V. =6/6; L. E. emmetropia, V. 6/6. R. E. +1.00 Sph. gives Jaeger 1 at 25 cm. L. E. +1.00 Sph. gives Jaeger 1 at 22 cm. The refraction was the same at this time with homatropin cycloplegia.

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[11] Knapp: Ztschr. f. Augenh., 1909, xxi, 420.

**Knapp's**[11] **Case 2.**—The patient was a man of 48, who had a severe diabetes of only a few weeks' duration when, in April, 1908, he complained of dimness of vision, coming on over night, for both far and near. Skiascopy revealed a hyperopia of 1 D., and +1.25 Sph. brought vision up from 1/3 to normal. To read Jaeger 1 at 20 cm., +5.00 Sph. was required, showing a weakened accommodation. Four weeks later the sugar had disappeared and the patient's sight was good without lenses. Skiascopy revealed emmetropia and V. =6/6 without lenses. Accommodation, 2 to 2.50 D. In March, 1909, emmetropia, both eyes. and V. =6/6. With +1.00 Sph. the patient read Jaeger 1 at 35 cm. He continued well and worked as a chemist without glasses. Neither of Knapp's cases had any abnormality of tension or demonstrable lesion in the eye.

**Zentmayer's**[12] **Case.**—The patient was a woman, aged 58, who had been under treatment for glycosuria for three weeks when she consulted the ophthalmologist, Oct. 4, 1909, complaining of rapid loss of vision for the past ten days. She had worn glasses only for near work during the preceding six years, but now could see better with these lenses for distance than without them. Vision was R. E.=6/60, L. E.=6/60; fields practically normal. Tension of left eye +?. There was marked arteriosclerosis of the retinal vessels. Otherwise the eyes were normal. Refraction was found to be:

R. E. +2.50 Sph. +0.25 Cyl. Ax. 165°, V. =5/5, L. E. +2.50 Sph. +0.50 Cyl. Ax. 15°, V. =5/5; +2.50 Sph. added, both eyes, enabled her to read type 0.37 at 25 cm. October 26, there was no change noted in the hyperopia or vision. November 9, R. E. +1.25 Sph. V. =6/5 pt.; L. E. +1.25 Sph. V. =6/5 pt. Accommodation unchanged.

Jan. 17, 1910, R. E. +0.50 Sph. V. =6/6 pt.; L. E. +0.75 Sph. V. =6/6 pt. Accommodation unchanged. Dr. Zentmayer states that the sugar, which had been as high as 5 per cent., was lessening at the time when vision failed, and had been absent for two days previous to her first visit to him (October 4).

**Roberts'**[13] **Case.**—Roberts reported the following case under the title of "Transient Cycloplegia Due to Glycosuria": A man, aged 51, the subject of diabetes, complained of inability to read with his old reading lenses and inability to see distant objects distinctly without lenses. Heretofore he had seen distinctly, without lenses for distance, and with +1.75 Sph., both eyes, for near. The sugar in the urine was now 3/8 per cent., whereas two weeks before it had been 7 per cent. R. E. V. =6/22.5; L. E. V. =6/15. Manifest refraction was: R. E. +1.75 Sph. V. =6/5; L. E. +1.75 Sph. V. =6/5. The addition of +2.25 Sph., both eyes, was necessary to bring the near point to 13 inches. No lesion was discovered in either eye. One month later his general health was improved. His last glasses had been satisfactory until within a few days; but now they blurred his distant vision, and required that he hold his book too close to the eyes. Vision, with correction, was 6/12. Manifest refraction showed: R. E. +1.00 Sph. V. = 6/5; L. E. +0.75 Sph. V. =6/5; with +2.25 Sph. added for near. Nine months later he reported that his eyes had gradually returned to their previous condition and he used his original lenses for reading (+1.75). It would seem to us that this is really a case of increase of hyperopia in the course of diabetes, with a very small amount of paresis of accommodation; for the patient required +1.75 Sph. for presbyopia before his diabetes, and only +2.25 Sph. added to his distance correction when first seen by Dr. Roberts; and when seen later his presbyopia remained the same, while his hyperopia had diminished 0.75 D. in the right eye and 1.00 D. in the left; later still both the hyperopia and the slight paresis of accommodation disappeared practically, allowing the use of the original reading lenses only (+1.75).

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[12] Zentmayer: Abstracted in Ophth. Rec., February, 1911.
[13] Roberts: Arch. Ophth., 1909, xxxviii, No. 4.

GOULD'S[14] CASE.—In April, 1907, Dr. George M. Gould reported a case of rapidly changing refraction in the person of a physician suffering from glycosuria, and reviewed the literature. His patient was a man, 58 years of age, whose eyes he had repeatedly examined under cycloplegia. In December, 1902, he had been wearing:

R. E. —2.62 Sph. —0.75 Cyl. Ax. 35°, V. =20/20+; L. E. —2.62 Sph. —1.25 Cyl. Ax. 167°, V. =20/20+, with +1.75 added for near in bifocals. On the eighteenth of that month the patient complained of eye-strain and blurred distant vision and accepted the following:

R. E. —3.25 Sph. —0.75 Cyl. Ax. 25°, V. =20/20+; L. E. —3.25 Sph. —1.50 Cyl. Ax. 166°, V. =20/20+, with +2.12 added for near in bifocals. There was also a limitation of the range of accommodation. Other symptoms suggested examination of the urine, which was found loaded with sugar. A strict diet was prescribed, and in a week the sugar had disappeared from the urine, but the eye-strain had returned. A reexamination showed that the error of refraction had returned almost exactly to the point of seven days before. In the next four years the patient was less careful of his diet, and in December, 1906, accepted the following:

R. E. —2.87 Sph. —0.37 Cyl. Ax. 25°, V. =20/20+; L. E. —3.00 Sph. —1.50 Cyl. Ax. 172°, V. =20/20+, with +2.50 added for near in bifocals. In February, 1907, there was increasing evidence of ill health, a high percentage of sugar in the urine, and symptoms of eye-strain. The rigid diet was resumed and in a few days the glycosuria had disappeared, but the eye-strain was worse than ever. Examination showed the following error:

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[14] Gould: New York Med. Rec., April 20, 1907.

R. E. —2.00 Sph. —0.62 Cyl. Ax. 20°, V. =20/20+; L. E. —1.87 Sph. —1.37 Cyl. Ax. 170°, V. =20/20+, with +2.00 added for near.

Dr. Gould writes us that the patient continues in good health and is now wearing:

R. E. —2.50 Sph. —0.87 Cyl. Ax. 35°, V. =20/20+; L. E. —2.50 Sph. —1.50 Cyl. Ax. 170°, V. =20/20+, with +2.37 added for near.

Attention is called to the fact that, ignoring the astigmatism, the myopia increased from —2.62 to —3.25 and then, after a period of careful dieting, fell to —2.00 in one eye, and —1.87 in the other, five-eighths of a diopter in one eye and three-quarters in the other, below what may be termed the normal myopia for the individual—the amount present before the glycosuric changes began. This, we believe, is not to be explained on the ground of careless refraction or lack of cycloplegia.

Dr. Gould very properly calls attention to the fact that no case of this sort can adequately be studied without cycloplegia if the patient still retains any accommodation, and that the previous static refraction should be known. He also says that there is no reported case of myopia (with sudden change in diabetes) in which glycosuria has not had the effect of increasing the myopia. He believes it illogical, if not impossible, that the same cause can also bring about an increase of hyperopia, and questions the accuracy of the observation of those who have reported such cases. It seems to us that Dr. Gould's own case disproves his contention. As we have already pointed out, after increasing and then falling back to normal, and increasing again, his patient's myopia diminished five-eighths and three-quarters of a diopter below the starting point. The following cases from our practice, however, show that the refraction may increase and diminish in the same patient in glycosuria:

WESCOTT'S CASE.—Miss K. H., stenographer, aged 36, was referred to one of us (C. D. W.), because of headache, in February, 1896. Under complete cycloplegia with homatropin and cocain she accepted:

R. E. +0.75 Sph. V. =6/6; L. E. +0.75 Sph. V. =6/6. She was given the full correction for near work only, with relief from her headaches. In May, 1898, she was complaining again and under homatropin and cocain accepted:

R. E. +0.87 Sph. V. =6/6; L. E. +0.62 Sph. V. =6/6. She was given the above, less 0.25 Sph., for constant use, and was again relieved of her symptoms. In May, 1903, she suffered again from headache and was again examined under homatropin and cocain, accepting:

R. E. +1.00 Sph. +0.25 Cyl. Ax. 180°, V. =6/6+; L. E. +1.25 Sph. +0.25 Cyl. Ax. 180°, V. =6/6+. She was given the above, less 0.37 Sph., and was comfortable once more. In July, 1906, she was again complaining, and under the same cycloplegia accepted:

R. E. +1.25 Sph. +0.25 Cyl. Ax. 180°, V. =6/6; L. E. +1.00 Sph. +0.25 Cyl. Ax. 180°, V. =6/6. Given the above, less 0.25, for distance and added +0.50 for near in bifocals. In September, 1907, when the patient was 47 years of age, she returned, saying that she could not see well with her distance glasses. The external eye, reflexes, and fundus were normal. Under homatropin and cocain she now accepted:

R. E. —0.25 Sph. +0.50 Cyl. Ax. 15°, V. =6/6—; L. E. —0.25 Sph. +0.50 Cyl. Ax. 165°, V. =6/6—. A few days later without cycloplegia she accepted:

R. E. —0.50 Cyl. Ax. 105°, V. =6/6—; L. E. —0.50 Cyl. Ax. 75°, V. =6/6—. This was prescribed, with +0.75 Sph. added in bifocals. There was no paralysis of accommodation. Diabetes was suspected and the patient was advised to consult her physician for general examination and treatment. November 2, 1907, she returned, complaining that she could no longer see to read with the glasses prescribed in September, and her bifocal segments were increased to +1.50 Sph. At this time her physician, Dr. Annie Sage, reported 5 per cent. of sugar in the urine. November 12, sugar 8.266 per cent. Acetone absent. March 19, 1907, the patient returned, complaining that she could not see well, far or near, with or without glasses. Examination showed that distant vision was reduced to 6/45, with either eye, with her correction, and near vision to Snellen 2 D. at 14 inches. With +3.00 Sph. added, distant vision was restored to 6/6—, and near vision to Snellen 0.50 D. at 8 to 18 inches. She was given +3.00 Sph. in a grab front to be worn over her bifocals. Sugar 4.533 per cent. Diacetic acid and acetone absent.

November 25, sugar 6 per cent.

December 2, sugar 4.266 per cent.

December 12, 1907, the patient returned her grab front, as the vision had improved so much that she was able to get along with her correction. Distant vision at this time was 6/12 with either eye. There was 5.866 per cent. of sugar in the urine on March 28, 1908; she saw better without her glasses than with them, and was again examined under homatropin and cocain, with the following result:

R. E. +1.00 Sph. +0.37 Cyl. Ax. 180°, V. =6/6; L. E. +1.00 Sph. +0.37 Cyl. Ax. 5°, V. =6/6. The patient was given the above, less 0.25 Sph., for distance, with +1.25 Sph. added in bifocals. At this time when the refraction had increased to and beyond what was normal for her when the rapid change began, the urine contained 6.66 per cent. of sugar. After this examination, however, her health failed rapidly, the amount of sugar increased to 15 per cent., and she died in coma, without being seen by us again. This case also shows that hyperopia may decrease, increase, and decrease again, while the amount of sugar more or less steadily increases.

Ellis' Case 1.—Catherine M., domestic, aged 40, seen in December, 1907, for some time had noticed that vision was variable, and three weeks previously Dr. N. S. Davis of Chicago had diagnosed diabetes. Vision: R. E. 6/30; L. E. 6/15. Under cycloplegia with homatropin and cocain she accepted the following:

R. E. +4.50 Sph. V. =6/6+; L. E. +4.50 Sph. V. =6/6+. A few days later, without cycloplegia, she accepted:

R. E. +4.00 Sph. V. =6/6+; L. E. +3.75 Sph. V. =6/6+, and this was ordered for constant use. At this time the urine contained 3 per cent. of sugar.

In March, 1908, the patient returned, complaining that she could no longer see well with the glasses and had discarded them. Reexamination under homatropin and cocain gave the following result:

R. E. +2.25 Sph. +0.25 Cyl. Ax. 10°, V. =6/6; L. E. +1.37 Sph. +0.37 Cyl. Ax. 30°, V. =6/6. Given the above, less +0.50 Sph. The patient was then passing four quarts of urine containing 2.5 per cent. of sugar.

Ellis' Case 2.—Mrs. J. T. W., aged 48, was referred by Dr. Julius Hoelscher of Chicago in September, 1910. The patient complained that her eyes had so changed in the past few weeks that she could see clearly in the distance with glasses used for some time for reading, and that they were no longer satisfactory for near work. Dr. Hoelscher reported polydipsia and polyuria and 4.5 per cent. of sugar in the urine. No diacetic acid or acetone was present. The patient had been examined by him one year before, and six months before, and no diabetes was found. The external eye, the reflexes, and the fundus were normal. R. V. =6/45, L. V. =6/45. With old reading glasses:

R. E. +2.50 Sph. +0.75 Cyl. Ax. 45°, V. =6/5; L. E. +3.00 Sph. V. =6/5. Under homatropin and cocain the patient accepted:

R. E. +2.75 Sph. +0.50 Cyl. Ax. 60°, V. =6/5; L. E. +2.75 Sph. V. =6/5. After passage of mydriatic a few days later she accepted:

R. E. +2.50 Sph. V. =6/5; L. E. +2.50 Sph. V. =6/5. The above was ordered, with +1.50 Sph. added for near. There was no paralysis of accommodation. In December, 1910, the patient came complaining of poor distant vision with glasses, and stated that vision was much better without them. Examination showed vision 6/9 in either eye without glasses, and 6/30 with correction. Without mydriatic she accepted:

R. E. +0.75 Sph. +0.50 Cyl. Ax. 45°, V. =6/5—; L. E. +0.75 Sph. V. =6/5, which was given, with +1.50 added for near. At this time the urine was free from sugar. February 14, 1911, this patient was again examined under homatropin and cocain. The fundus was normal and she accepted:

R. E. +1.00 Sph. +0.37 Cyl. Ax. 45°, V. =6/6+3; L. E. +1.25 Sph. +0.25 Cyl. Ax. 165°, V. =6/6+4. A few days later without mydriatic she accepted the above, less 0.25, and +1.50 added for near. No paralysis of accommodation. This case, like our first one, shows an increase of hyperopia, though we do not know how much, followed by a very marked decrease.

Ellis' Case 3.—A woman, aged 65, was seen July 12, 1910, Her distant vision had been failing rapidly for three months, with dull headache on near work. The ophthalmoscope showed sclerosis of the lenses and pronounced retinal arteriosclerosis. R. V. =6/22—1, L. V. =6/22—1. She was wearing +2.75 Sph., both eyes, for near, and read Snellen 0.50 at 9 inches. Under homatropin and cocain she accepted:

R. E. —0.75 Sph. —1.00 Cyl. Ax. 15°, V. =6/9; L. E. —0.75 Sph. —0.50 Cyl. Ax. 135°, V. =6/12. She was given the above, with +2.50 added for near. On August 16, 1910, she saw better without glasses. R. E. V. =6/22; L. E. V. =6/22; with glasses V. R. E. =6/45; L. E. =3/45. Under homatropin and cocain she accepted:

R. E. +1.75 Sph. +0.25 Cyl. Ax. 120°, V. =6/9+; L. E. +1.75 Sph. +0.25 Cyl. Ax. 180°, V. =6/9. March 17, 1911, she again saw better without distance glasses than with them. She admitted carelessness in diet.

R. V. =6/12+, with correction =6/30; L. V. =6/12+, with correction =6/30. Under homatropin and cocain she accepted:

R. E. +0.75 Cyl. Ax. 130°, V. =6/9+2; L. E. +0.50 Cyl. Ax. 15°, V. =6/9—2.

A review of the above cases shows that a much more extended and accurate study is necessary for a solution of the problem involved. As has been pointed out, the static refraction of the patient in health should be known, and careful examinations are desirable as often, at least, as the patient is able to recognize changes in his vision. It goes without saying that all examinations should be made under cycloplegia when any accommodation remains which could interfere with accuracy. Frequent and regular examinations of the urine should be made for sugar and other substances which may affect the refraction or the accommodation. The fact that there is no definite relation between the amount of sugar in the urine and the refraction changes is very suggestive, as is also the fact that partial or complete paralysis of accommodation may or may not be present in cases showing an equal amount of sugar in the urine. The fact that the most startling changes in the vision and refraction have occurred when the general condition was improving and the urine free from sugar, points to the possibility of another agent as the offending factor in at least a portion of the cases. We regret exceedingly not being able to make more extended studies in our own cases; but two of our patients were working women who could give us little time, and who gave the internist even less opportunity for the study of their metabolism.

Numerous theories have been proposed in explanation of refraction changes in the course of diabetes, generally assuming that the presence of sugar in the lymphatic circulation of the eye produces alteration in the refractive indices of the media, cornea, aqueous, lens, and vitreous; or else that the volume of the globe is altered through the loss of fluid in diabetes. For example, Landolt[6] explained his case of diminished refraction on the theory of augmentation of the index of refraction of the vitreous body, since the vitreous presents a concave surface to incident rays. He thought that changes in the volume of the globe could not occur rapidly enough to account for the changes in refraction observed in his patient. On the contrary, Horner[1] believed that sudden loss of fluid from the eye was responsible for developing hyperopia. Schmidt-Rimpler,[15] not referring to diabetic cases especially, said that loss of fluid, with minus tension, does not alter the refraction of an eye. In perhaps the most thoroughly studied of all the cases found in the literature, Lundsgaard's third case,[10] repeated examinations with the ophthalmometer and the Schiötz tonometer revealed no alterations in corneal curvature or tension of the eyes during the period of varying refraction.

An increase of refraction in the course of cataract development is a very frequent phenomenon. On the contrary, Machek[16] detailed three cases of diminished refraction in the course of the devolepment of subcapsular cataract with central transparency of the lens; and Priestley Smith[17] speaks of hyperopia developing in incipient cataract. In diabetic cataracts, the lens has been found, on chemical examination, to contain sugar

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15. Schmidt-Rimpler: Graefe-Saemisch Handbuch der gesamten Augenheilkunde 6, vii.
16. Ztschr. f. Augenh., xix, 330.
17. Smith, Priestley: Graefe-Saemisch Handbuch des gesamten Augenheilkunde, 11, vi.

(Liebreich, Leber); Nagel, on the contrary, did not find sugar in the lens. Lichtenstein[5] thought that change in the index of refraction of the lens was the explanation of his case, as did Knapp,[11] who compared the diabetic toxin with nicotin and strychnin in the rapidity of its action.

Against the idea of alteration in the refractive index of the aqueous caused by the presence of sugar in this fluid, is the experiment of Hess[18] showing that 20 per cent. of grape-sugar is necessary to produce a myopia of 1 D., while Deutschmann[18] noted cloudiness and swelling of a lens after immersing it in a 5 per cent. sugar solution for a few hours. Obviously these two experiments are inconsistent with the idea that a considerable change in refraction, without cataract formation, is due to the presence of sugar in the fluid bathing the lens.

It seems to us that none of these theories will do. The problem is an interesting one, however, and no doubt will some day be solved. It offers opportunity for the internist, the physicist, and the physiologic chemist, as well as the ophthalmologist.

In conclusion, we wish to express our indebtedness to Dr. Emory Hill for his painstaking care in abstracting the published cases, and for other assistance in the preparation of this paper.

31 Washington Street.

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18. Hess: Quoted by Parsons: The Pathology of the Eye, iii, 929.

## MEMORANDA

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# THE PSYCHOLOGIC ASPECT OF REFRACTION *

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While refraction is a science based on mathematical principles and theoretically very exact, yet there are other questions involved besides that of measuring the radius of curvature, finding the far and near points, range of accommodation, etc.

What I shall have to say in this short paper will not be new to many, but it is a phase of refraction about which very little has been said or written. Hence my excuse for presenting this paper.

Has psychology any practical bearing on the subject of refraction? In many cases perhaps very little, while in other cases quite a good deal. If all brains were of the same quality, all minds trained to think alike, all nervous systems equally well balanced, every individual reared in a pure atmosphere with proper and healthful environments, refraction would in a measure be reduced to the simple question of accurate observation and correct measurements. It would be largely mechanical. But as such conditions do not and cannot exist, we are bound to recognize the right of psychology to a place in refraction. We must in every case consider the individual with all his idiosyncrasies and eccentricities. Refraction to the beginner is a fascinating subject. It appeals to him because of its exactness. It presents problems which he can work out for himself, and he can prove the correctness of his conclusions. He is therefore liable to underestimate the gravity of the subject, and to overlook one of the most vital features of it; that is, the individuality of the patient, as well as that of the

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examiner. I shall not attempt to go into the philosophy of mental suggestion or to discuss the various theories of psychic phenomena, because it is not necessary or expedient to do so, and the field is too broad.

We are compelled to admit that there is a psychologic phase to every situation in which two minds are brought in close relationship; where the personality of the one appeals to that of the other, and where a motive exists for the obtaining of a sympathetic relationship between the two. To have a strong personality, or to be possessed of what is usually termed personal magnetism, is a very necessary element of success in any calling in life, and it is worth as much to the refractionist as to any other. In fact, the refractionist who does not possess a reasonable amount of the faculty is handicapped.

The fitting of glasses to presbyopes, in a large percentage of cases, does not call for any great skill or an extensive knowledge of scientific refraction, but does require a reasonable amount of common sense and a good knowledge of human nature. These patients are not hard to please, as a rule, and are satisfied with vision which enables them to read ordinary print without much effort. This assertion applies, of course, to the rank and file of those who have accepted the portion of senility, who have worn off the wire edge of enjoyment and have resigned themselves to the simple life. I have seen patients with decided astigmatism plodding along contentedly with a very indifferent pair of spherical lenses, in frames set all askew. A person of this class of humanity is easily imposed on by the unscrupulous spectacle-vender, and can usually fit himself if you will furnish him with a few pairs of glasses from which to select. There are others, however, who have very decided ideas of the dangers which may come from the wearing of glasses incorrectly fitted and who would be very unhappy if they had been fitted by one in whom they had not the utmost confidence. And still, they may not be unreasonable in their requirements and need only to be assured that they have had a proper refraction and that their glasses are correct.

There are others, however, who are skeptical and disposed to find fault. They are never quite sure of anything; are afraid to admit that they see any better with one lens than another, lest they make a mistake and mislead the refractionist. They will hesitate as to the relative clearness of two lenses, trying first one, then another; then they will go back to the first again, and so seesaw back and forth until the refractionist has exhausted his stock of patience and nervous energy, and the patient is in such a state of uncertainty that he cannot render any intelligent assistance. Perhaps the most sensible thing to do in this case would be the postponement of the work to another day. This gives the refractionist time to take the measure of this patient and plan his campaign, when, perhaps, by bringing to bear a large amount of his own personality the patient may be made to accept a correction. A patient of this class is likely to give the physician more or less trouble later, for having a doubting disposition he will do some testing on his own account and return from time to time with his grievances. Whether the grievance be real or otherwise does not much matter, for he thinks it is a real one and the case must be given some attention. If the refractionist feels sure that he has given a proper correction, then it becomes a question of dealing with the mental attitude of the patient. Confidence must be established and ghosts of imaginary evil and preconceived ideas of dire calamity must be dissipated. With this class of patients there is a fertile field for the exercise of a strong personality and psychologic influence.

Much more might be said along this line, but I wish to discuss briefly another phase of humanity, one of the most troublesome types with which we have to deal; i. e., the man (or woman) who thinks that glasses would in some way or other be an advantage to him. He has diagnosed his own case by introspection or reading quack advertisements, or else he thinks that glasses will lend a certain appearance of respectability or intellectuality, and insists on having them. The individual of this particular type is usually ignorant and could get on just as well without glasses as with them, even if he should happen to have a small amount of ametropia, but he wants them and will very frequently deceive the examiner—unintentionally, perhaps. His anxiety to obtain the glasses will lead him to say that he sees better when he really does not, and nearly any lens placed before him is good. His replies cannot be depended on at all as to how well he sees. The only sure test is to make him call the letters, if he knows them. Sometimes, however, he will say that a certain lens is an improvement, not from any intent to deceive, but because he is led on by mental suggestion to say that he sees better or worse. He is impressed with the idea that he is expected to see better, and answers the question accordingly, but when called on to read the letters he stumbles as badly or even worse than before.

While I do not approve of suggestive therapeutics or hypnotism as practiced by the ill-advised or the charlatan, I do recognize that much can be done with individuals of this type by mental suggestion. They have very plastic, impressionable minds. They are credulous and more or less superstitious and easily imposed on, and are the legitimate prey of the unscrupulous. Still this same inexplainable something which we call personality, this power to impress and inspire confidence, is a very potent one, and the refractionist who is lacking in it will experience great difficulty in leading the patients along lines of least resistance to correct conclusions. It is also necessary to be a good judge of human nature, to be able to interpret mental attitudes of a patient, to recognize elements of hysteria and detect symptoms of remote reflexes and neuroses, to be able to say to your patient that, with the restoration to a normal condition mentally and physically, all eye symptoms will pass away. More than this, it is necessary to carry conviction to the mind of the patient and furnish a substitute for all his preconceived ideas. Sometimes it seems advisable to compromise with him and give glasses when one is convinced that there is no real need for them. Some of these patients come to recognize in time that they get on just as well without their glasses, and lay them aside, while others continue to wear even plain glasses with a great deal of confidence and will stoutly maintain that their eyes soon grow tired and give trouble when the glasses have been left off.

So the glasses have served a good purpose and the end has justified the means in a measure. With the conscientious physician the bringing to bear of all of these faculties and forces, if the interest of his patient be subserved, is a perfectly justifiable procedure; it is honorable and legitimate. But with a refractionist who is looking only to the monetary side of the question, or with the quack spectacle-vender, whose mission is to sell glasses regardless of the requirements of the case, the exercise of this faculty becomes a means of perpetrating fraud, and cannot be too severely condemned.

I recall the case of an optometrist hailing from New York, who rented the front rooms on the parlor floor of the leading hotel of the city and advertised the most marvelous results from his prescription lenses and methods. He had patients flocking to him from all over the district, to be treated for all manner of diseases. He was treating corneal ulcers, granular lids, cataracts, optic atrophy, paralysis agitans, nystagmus, etc., promising cures in all cases, and collecting fees from ten dollars up to one hundred and fifty, as well as getting the indorsement of some of the most prominent people of the community. His stock-in-trade was skilful advertising, a wise and mysterious air and a spectacular way of doing business, which fairly took the people off their feet. The man was an artist in his way.

I recall another instance of a similar character, in which a traveling optician rented the parlors of the leading hotel of the city and for two months did a business which was amazing. He handled only blue-tinted lenses, and sold to nearly every one who went to him two pairs of spectacles. For two pairs of gold-framed spectacles he got thirty dollars; for two pair of steel spectacles fifteen dollars. His glasses were supposed to possess some marvelous chemical properties for the curing of all manner of eye troubles.

Both of these men made the mistake of coming back when the people had had time to think a bit, and see that they had been faked. The psychologic influence was gone, the spell was broken, the hold on the community lost, and the visit a failure.

It is right and proper for the ophthalmologist to keep himself well equipped, to supply himself with the most improved instruments. It is commendable to surround himself with all the paraphernalia possible to present an appearance of respectability and to seek the confidence of his patients, to bring them into his way of thinking, provided these things are done in the interest of the patient; but to use the arts and wiles of the hypnotist or to indulge in the use of questionable measures which are calculated to exercise undue influence over the patient or deceive him for the purpose of getting a fee or selling him some glasses, is dishonorable. Still, this tragedy is being enacted every day, and the sad part of it is that we seem to have no recourse. Any effort to protect the interest of the people is looked on as an act of jealousy. One's motives are sure to be misinterpreted, and his kind offices neglected by the patient, while the faker makes capital of what he terms persecution on the part of the regular physician.

An effort was made by the State Medical Association of Alabama at the last session of the legislature to get a bill through which would enlarge the power of the state board so that advertising quacks could be more satisfactorily dealt with, but the measure was fought so energetically by the quacks and newspapers of the state that the friends of the bill were forced to amend it, and provide for trials by jury. The original bill called for the trial of all unprofessional conduct and extravagant advertising, before the state board, and the measure would have carried, even in the face of the opposition outside of the state association, but for a fight on the part of some of the members for the right of trial by jury.

## MEMORANDA

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# NYSTAGMUS *

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At the suggestion of one of the honored older members of this section, I have undertaken to discuss the subject of nystagmus as concisely and as simply as possible.

The term "nystagmus" is used to denote jerkings of the eyes which do not affect the usual voluntary eye movements incidental to visual acts, and in my opinion, should be applied to those jerkings only which possess uniformity of character and direction. All other eye jerkings, such as the aimless variety seen in certain blind eyes and the eye tremors of hysteria and multiple sclerosis, I regard as false nystagmus. Nystagmus is a physiologic response to certain tests under normal conditions, and a symptom of certain abnormal conditions.

## PHYSIOLOGIC NYSTAGMUS

It is misleading to call the right labyrinth or the left labyrinth by the term "vestibular apparatus." The human body has only one "vestibular apparatus"—which comprises two labyrinths, two vestibulo-cerebellar tracts connecting the labyrinth of each side with the cerebellum of the same side, and two vestibulo-ocular tracts which connect the labyrinth of each side with the vestibulo-ocular center of the same side. Each vestibulo-ocular center has direct nerve connections with the extra-ocular muscles of both eyes, these nerve connections being such that stimuli emanating from one vestibulo-ocular center cause contractions of the adductor of the same side and of the abductor of the opposite side.

The vestibular apparatus may be looked on as a physiologic seismograph with additional mechanism permit-

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ting recognition of position in space with respect to the force of gravity. This apparatus depends for proper functionation on normal activity in each of two labyrinths, one situated on the right side, one on the left. Each labyrinth comprises a common utriculus and three semicircular canals at right angles to each other in the three cardinal planes of space, both ends of each canal opening into the utriculus. Immediately adjacent to the utriculus on one end of each canal is found an ampulla, within which ciliform sensory end-organs project into the endolymph which fills utriculus and canals. In quiescence these cilia project directly upright into the lumen of the semicircular canal, inclining neither toward nor away from the utriculus. Movements of these cilia by currents of endolymph institute nerve impulses, the character and intensity of which are normally such as to maintain a state of vestibular equilibrium between the two labyrinths. When vestibular equilibrium is upset, the character of the disturbance depends on whether the cilia bend toward or away from the utriculus. The effect of changed position of the cilia differs in the different canals. Preponderance of either labyrinth is created by causing the cilia of the superior semicircular canal of that labyrinth to point away from the utriculus or by causing the cilia of the horizontal semicircular canal of that labyrinth to point toward the utriculus. We are ignorant concerning the result of changes of ciliary position in the posterior semicircular canal.

Inasmuch as equilibrium or balance is only relative, it makes no difference whether equilibrium is upset by absolute increase of one side, or by diminution of influence of the other side. For instance, the normal vestibular apparatus may be compared to a pair of scales in equilibrium with a normal of 10 pounds on each side. The left side of the scales will preponderate when a weight of 5 pounds is added to the left, or when the same weight is taken away from the right. Preponderance of one labyrinth is always relative. Stimulation of one side is comparable to an increase of the weight in the above simile; depression of one side is comparable to lessening the weight. Balance being upset, it matters not whether by depression of one labyrinth or stimulation of the other, there results a slow involuntary excursion of the eyes toward the side opposite the preponderating labyrinth. As soon as the gaze is thus withdrawn from a desired object, the cortical impulse redirects the gaze, causing a quick return of the eyes in the opposite direction in search of the object of fixation. Fixation no sooner occurs than the reflex stimulus again prevails, instituting another slow excursion, which is followed by a quick return. Thus is set up a series of rhythmic eye jerkings which constitute physiologic or reflex nystagmus.

Nystagmus of this type is the result of antagonistic central impulses, the involuntary impulse from the vestibular apparatus causing an excursion in one direction, the voluntary impulse from the sensorium causing a quick return in the other direction. If the gaze be directed voluntarily toward the same side to which the involuntary impulse directs it, the nystagmus disappears, and conversely, if the gaze be directed voluntarily toward the opposite side, the maximum of nystagmus will be elicited.

Disturbing vestibular equilibrium by stimulating a single canal on one side, whether at the same time depressing its fellow of the opposite side or not, causes nystagmus in the same plane as the canals, toward the side opposite the stimulated canal; for example, stimulation of the left superior canal or depression of the right superior canal, or both, causes rotatory nystagmus to the right; or, depression of the left horizontal canal and stimulation of the right horizontal canal causes horizontal nystagmus to the left. Stimulation of all the canals of one side simultaneously always causes rotatory nystagmus toward the opposite side.

Reflex nystagmus is best elicited by so changing the temperature of the endolymph by the use of a stream of cold water in the external auditory canal as to cause a downward current of endolymph in whichever canal is upright, viz., when the head is bent forward, in the horizontal canal, when the head is in upright position, in the superior canal. By using a double cold water tube, one side may be examined separately, or both sides simultaneously, as the physician may desire.

Rotation of the patient on a vertical axis turning-stool is a very convenient and practical means of eliciting nystagmus. By changing the posture of the head the planes of the different semicircular canals are brought to right angles with the axis of rotation, thus allowing examination of the different canals. Rotation experiments affect both right and left labyrinths simultaneously—and oppositely (i. e., depressing the one side when stimulating the other side) except in the case of the posterior canals, which are affected simultaneously and similarly by rotation, because their ampullæ are not oppositely placed, as are the ampullæ of the superior and horizontal canals. Rotation should always be made by ten revolutions in the direction of the movement of clockhands. After rotation with the head in the upright position the left labyrinth will be stimulated and the right labyrinth will be depressed, causing rotatory nystagmus to the left; after rotation with head bent backwards, the right labyrinth will be stimulated and the left depressed, causing rotatory nystagmus to the right. After rotation with head tipped over either shoulder the posterior canals of both labyrinths would be affected, causing vertical nystagmus.

Electric influence may be used to elicit reflex nystagmus. The cathode applied to the ear or mastoid stimulates the activity of the labyrinth of that side. The anodal influence is the reverse.

Reflex nystagmus always causes apparent movement of fixed objects.

Nystagmus takes its name from the quick component; for instance, after a slow excursion toward the left, comes a quick return toward the right—"nystagmus to the right."

### NYSTAGMUS AS A SYMPTOM

Two kinds of nystagmus are found as symptoms; rhythmic, consisting of a slow component and a quick component; and undulating, consisting of excursions to and fro of equal velocity and extent, like the swing of a pendulum. Rhythmic includes the "vestibular" and the "retrovestibular" or "cerebellar" types of nystagmus; undulating, the so-called "ocular" type.

*Vestibular Type.*—Circumscribed labyrinthine disease acts similarly to any form of irritation on the affected labyrinth, stimulating its activity. The normal reflex from such stimulation, granted the vestibular tissues are capable of functionating, takes the form of rotatory rhythmic nystagmus toward the diseased side. As disease progresses, more and more of the essential labyrinthine tissues become destroyed, impairing reactive ability, which impairment is evidenced by gradual diminution in the intensity of the nystagmus. By the time the stage of total labyrinthine destruction is reached, all nystagmus toward the diseased side will have disappeared, and rhythmic rotatory nystagmus toward the opposite side will have replaced it, because, the impulses from the destroyed labyrinth having ceased, normal impulses from the non-diseased opposite labyrinth preponderate. Profound deafness on the diseased side is a very early symptom of such cases.

*Retrovestibular Type.*—Certain diseases in the cerebellum effect a stimulation of the labyrinth of the same side, through the medium of the cerebello-vestibular tract, and find expression in rotatory rhythmic nystagmus toward the diseased side. As the disease progresses and the stimulation becomes greater and greater, the nystagmus increases in intensity, inasmuch as the vestibular tissues have suffered no impairment of functionating ability. In retrolabyrinthine or cerebellar disease, no deafness is found. It is, therefore, very easy to diagnose between nystagmus of the vestibular type and of the retrovestibular or cerebellar type. Both of these types of nystagmus always cause apparent movement of fixed objects.

*Ocular Type.*—I have evolved the following theory concerning the development of nystagmus of the ocular type: The extra-ocular muscles are controlled by proper functionation of the oculo-motor centers, which control is developed in the considerable period of time during which the infant is learning the visual act. A moderate degree of visual acuity is necessary to the correct learning of the visual act. Objects at first accorded but intentionless glances come to be given closer and closer observation as cerebral development proceeds, and voluntary control of the extra-ocular muscles is indispensable to such close observation. The meaningless wanderings of the eyes apparent during early life become replaced by associated movements of the head and eyes consistent with the development of the faculty of intention. The cortical impulse is a controlling one and must be regarded, therefore, as in a sense inhibitory. Learning goes on simultaneously through the normal channels of all the senses. The attentions of the child are distributed over the realms of sight, touch, hearing, tasting and smelling. Should visual acuity be sufficiently below normal, that share of the child's attentions which would normally fall to learning the visual act, will be diverted into some other channels, and the development of the normal visual mechanism and function will suffer by reason of neglect. As cerebral development proceeds cerebral activities increase, and the increasing activity in the oculomotor centers, not being checked by inhibitory cortical impulses, finds expression in nystagmus of the ocular type.

This nystagmus is undulating, uninfluenced by the usual voluntary eye movements, does not cause apparent movement of fixed objects, and is always associated with low visual acuity. Ocular type of nystagmus is seen in connection with amblyopia, albinism, excessive hyperopia, congenital cataract, hydrocephalus, macula or leukoma corneæ, anterior polar cataract, strabismus and pigmentary degeneration of the retina.

### FALSE NYSTAGMUS

In certain hysterical cases, irregular eye tremors are seen. In multiple sclerosis the extra-ocular muscles are the subjects of ataxic movements, or of intention tremor, or of both. These show no regularity either in movement or direction, and are easily distinguished from true nystagmus. They always cause apparent movements of fixed objects. The so-called miner's nystagmus is a habit spasm or neurosis resulting in vertical undulating or irregular movements of the eye-balls.

1130 Main Street.

# MEMORANDA

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# THE OCULAR CONJUGATE AND FUSION BRAIN CENTERS

## ONLY TWO AXES OF OCULAR ROTATIONS, AND ONLY TWO PLANES OF REFERENCE *

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In my investigations, some years ago, I saw the importance of finding a larger number of brain centers for the control of the ocular muscles than had been given in any list of these centers published up to that time. I not only found that there must be conjugate centers whose existence had never been announced, but that there must also be fusion centers. To the old list of conjugate centers I added six additional members; the sixth, the seventh, the eighth, the ninth, the tenth and the eleventh. The first, second, third, fourth and fifth conjugate centers had already been recognized, and Maddox had said "probably more." In addition to the six conjugate centers added to the original number, I found that there must be a fusion center for every individual extrinsic muscle, and also an individual center for each ciliary muscle and for each sphincter of the iris. In this paper I shall not refer again to the individual centers for each ciliary muscle and each sphincter of the iris, or to their conjugate centers; but only to the conjugate and individual centers pertaining to the extrinsic ocular muscles, nine of the former and twelve of the latter. I named the first eight of these fusion centers, right and left first basal centers, right and left second basal centers, right and left third basal centers, and right and left fourth basal centers. Notwithstanding it had been previously published that there was a fifth conjugate center, I found no evidence of the existence of right and left fifth basal centers, hence the skip from fourth to sixth. The remaining four I named right and left sixth and right and left seventh basal centers. To all of these basal centers, thus enumerated, I gave the general name of fusion centers, and declared them to be under the control of the fusion faculty of the mind. The purpose of this paper is to prove the existence of the nine conjugate centers connected with the extrinsic muscles of the eye, and the twelve fusion centers connected with these muscles. All of these are shown in Figure 1.

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I did not recognize at that time that all ocular rotations, whether monocular or binocular, were effected around only two axes. At that time it was a matter of general knowledge that rotations in the horizontal plane were around a vertical fixed axis, and that the vertical rotations were around a horizontal fixed axis. I, at that time, found myself in agreement with the teaching of those who had preceded me, that every oblique rotation was around a fixed axis and that this axis was at right angles to the rotation plane; but this is not true.

In Figure 2 I represent the equator of the eye and the vertical and transverse axes lying in the plane of the equator, and I unhesitatingly declare that around these two axes are effected all oblique rotations, and that throughout such a rotation each of these two axes is itself in motion; there are no fixed axes for oblique rotations.

I was not alone in the thought that an oblique point in the upper field might be reached by a rotation, first around the vertical axis and then upward on the transverse axis of the eye, without any torsion of the eye on reaching the obliquely placed second point of view. I was at that time, as I am now, convinced that in oblique rotations there was no torsion and I concluded that the torsioning did not occur because of the contraction of the obliques for the purpose of preventing it. I still believe, and shall prove, in connection with the study of Figure 3, that oblique secondary points of view are reached without torsion, and that this is prevented by the obliques.

A few weeks ago the thought occurred to me that, if two simple rotations, the first around the vertical axis and the second around the transverse axis, could enable the eye to reach the second point of view obliquely placed, without torsioning, the same muscles could effect

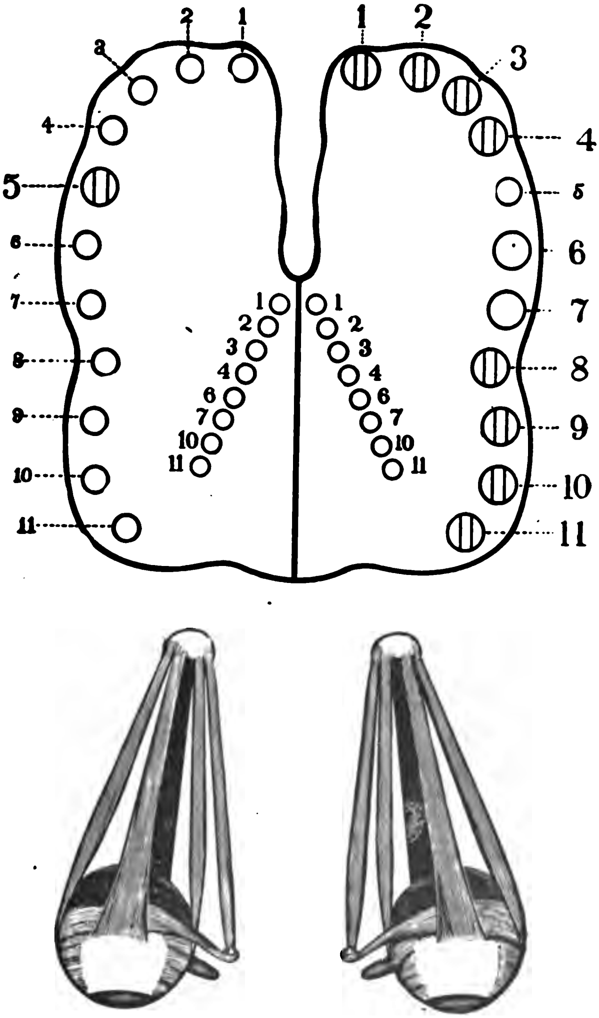

Fig. 1.—Conjugate and fusion brain centers.

the rotation in an oblique plane by acting simultaneously on the eye, one rectus effecting the upward or downward and the other rectus the right or left movement, while an oblique stands guard against torsion.

I at once appealed to my muscle indicator for proof of the correctness of the thought. I saw at once that, while the elevators were rotating the eyes on their horizontal axes, the lateral recti could rotate the eyes at the same moment around the vertical axes, and that the result of this double rotation taking place simultaneously would be the same as if the eyes had moved first around one of these axes and then around the other. I elevated the isogonal circle of the muscle indicator, carrying the visual axes with it, but at the same time, by another force, I shifted the axes in the plane of the isogonal circle and reached a secondary point of view without torsioning. The two forces were independent of each other, and yet were acting in most perfect harmony. Thus it is that I shall be able to prove, as I proceed, that every oblique rotation is effected by the conjoined work of three conjugate centers and two fusion centers, the five acting on three pairs of muscles; that one of these pairs elevates or depresses the two eyes, another pair shifts the visual axes to the right or left, as the case may be, and the third pair prevents any rotation around the visual axes; that one fusion center acts on one lateral rectus, hindered by an oblique, while the other fusion center acts, as the case may be, on one superior or one inferior rectus, hindered by an oblique.

That the study of Figure 3, in proof of the existence of all of the nerve centers named above, may be made easier, I shall group the twelve ocular muscles into the nine pairs, and in connection with each pair, shall name the conjugate center in control.

1. The two superior recti, under the control of the first conjugate center, for elevating the visual axes.
2. The two inferior recti, under the control of the second conjugate center, for depressing the visual axes.
3. The two interni, under the control of the third conjugate center, for the converging of the visual axes.
4. The right externus and the left internus, under control of the fourth conjugate center, for effecting rotations to the right.
5. The left externus and the right internus, under control of the fifth conjugate center, for effecting rotations to the left.
6. The two superior obliques, under the control of the sixth conjugate center, called into action only when the rotation is directly down and in conjunction with the second conjugate

**center and the two inferior recti, the purpose being to prevent outward torsioning.**

**7. The two inferior obliques, under control of the seventh conjugate center, but called into action only when the rotation is directly up, and then in association with the first conjugate center and the superior recti, the purpose being to prevent torsioning inward.**

**8. The superior oblique of the right eye and the inferior oblique of the left eye, under control of the eighth conjugate center and called into action only when rotations are obliquely up and to the right or down and to the left, to prevent torsioning to the right.**

**9. The superior oblique of the left eye and the inferior oblique of the right eye, under control of the ninth conjugate center, called into action only when the rotation is up and to the left, or down and to the right, to prevent torsioning to the left.**

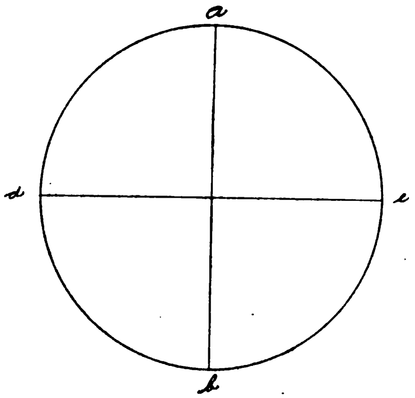

**Fig. 2.—The equator and the two axes of rotations.**

As to the fusion or basal centers, which could also be called the duction centers, the following is true:

The right and left first fusion centers belong, respectively, to the right and left superior recti. The right and left second fusion centers belong, respectively, to the right and left inferior recti. The right and left third fusion centers belong, respectively, to the right and left interni. The right and left fourth fusion centers belong, respectively, to the right and left externi. The right and left sixth fusion centers belong, respectively, to the right and left superior obliques. The right and left seventh fusion centers belong, respectively, to the right and left inferior obliques.

Having named all of the conjugate and fusion centers and the pairs of muscles, or individual muscles, to which they respectively belong, it now remains for me to prove, by means of Figure 3, the actual existence of all these. The two planes to which I wish to call special attention are the plane *a-b-c-d*, which represents the horizontal fixed plane of the head extended; and the plane *g-h-i-j*, which represents the vertical fixed plane of the head. These two planes, at right angles to each other, have their line of intersection in *e-f*. Points in space located anywhere on this line are primary points of view, and are known to be on this line of intersection. The plane *a-b-c-d* is a plane of reference so far as objects above or below it are concerned. The plane *g-h-i-j* is a plane of reference for all objects either to the right or left of this plane.

If *f* be the point of fixation and the lateral recti muscles are normal, only the third conjugate center has acted and only the two interni have responded. If the eyes are esophoric, to prevent the visual axes crossing between *e* and *f*, the right and left fourth basal centers must each act on its individual externus. If the eyes are exophoric, the right and left third basal centers must be called into action to prevent the crossing of the visual axes at some point beyond *f*. The neuricity from the right and left third basal centers has supplemented the neuricity from the third conjugate center.

If the eyes are to be rotated from *f* to *f″*, and are orthophoric, the third conjugate center will keep up the convergence and the fourth conjugate center alone, acting on the right externus and the left internus, will sweep the point of convergence from *f* to *f″*. If the eyes are esophoric the neuricity to the left internus would make the eye move faster and farther than the right externus would move its eye, if there were no supplementary supply for the latter muscle. This supplemental neuricity would come from the right fourth fusion center. If the eyes were exophoric, the neuricity from the fourth conjugate center would make the right eye move faster and farther than would the left, unless supplemental neuricity should be sent to the left internus. This supplemental power would come from the left third basal center.

If the eyes were to be rotated from *f* to *f′*, and they are orthophoric, the convergence would be kept up by the third conjugate center, and the point of fixation would be carried from *f* to *f′* by neuricity from the fifth conjugate center only, acting equally on the left externus and the right internus. If the eyes to be thus rotated are esophoric, the neuricity from the fifth conjugate center to the right internus would make the right eye move further and faster than that sent to the left externus would turn the left eye. To prevent this lagging behind of the left eye supplemental neuricity must come from the left fourth fusion center to the left externus. If such eyes were exophoric, the right eye would lag behind the left, unless supplemental neuricity should come from the right third basal center.

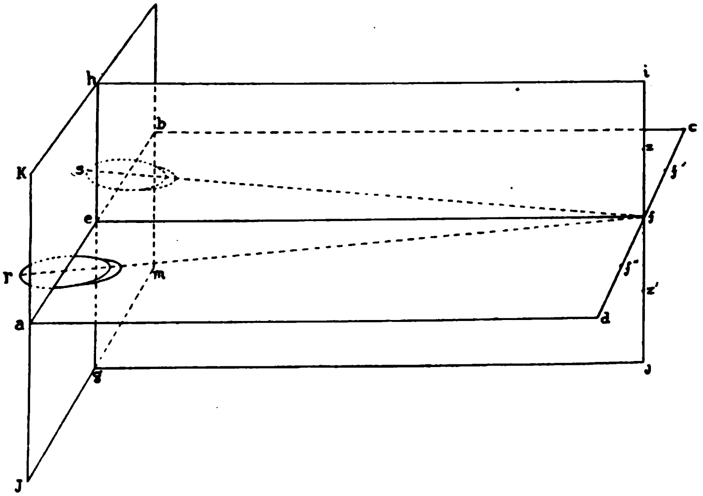

Fig. 3.—The two planes of reference.

If the rotation is to be from *f* to *z*, the visual axes must be lifted by the two superior recti, and the vertical axes must be kept parallel by simultaneous action on the part of the inferior obliques. The impulse to the former muscle would come from the first conjugate center, and that to the latter would come from the seventh conjugate center. If the eyes to be thus rotated are orthophoric, no basal center would be called into action, but if the left eye should be hyperphoric, the neuricity from the first conjugate center would make that eye move upward faster than the right eye would go, unless the superior rectus of the right eye should receive supplemental neuricity from the right first basal center. Reversing the hyperphoria, the neuricity would have to come from the left first basal center in order to make that eye sweep in harmony with its fellow.

Rotations of these eyes from *f* to *z′*, if orthophoric, would be effected by neuricity from the second conjugate center, acting on the two inferior recti to pull the visual axes down, and, simultaneously, neuricity would come from the sixth conjugate center to the two superior obliques, and no basal center would be aroused into activity. But, if the right eye be cataphoric, supplemental neuricity would have to be sent from the left second basal center to the inferior rectus of the left eye, in order to make it keep pace with its fellow in rotation. In either of these vertical rotations the convergence would be effected by the third conjugate center.

In any rotation above the horizontal plane, whether cardinal or oblique, the first conjugate center must be active. In any rotation below the horizontal plane the second conjugate center must be active. In any rotation to the right of the vertical plane, whether in the horizontal plane or above or below it, the fourth conjugate center must be active. In any rotation to the left of the vertical plane, whether in, above or below the horizontal plane, the fifth conjugate center must be active. In a cardinal rotation above the horizontal plane, the two inferior obliques must act together. In a cardinal rotation below the horizontal plane, the two superior obliques must act together. In oblique rotations up and to the right and down and to the left, the eighth conjugate center must act on the superior oblique of the right eye and the inferior oblique of the left. In any oblique rotation up and to the left or down and to the right, the ninth conjugate center must act on the left superior and the right inferior obliques. These oblique rotations, even of orthophoric eyes, cannot take place under the influence of the conjugate centers alone, for the reason that one oblique will help one of the recti muscles of a pair and hinder the other, and *vice versa,* of each eye, thus making it necessary for one basal center for a rectus muscle belonging to one eye to be active to overcome the retarding influence of the oblique muscle, and the same is true of the retarded rectus muscle of the other eye. To illustrate: In the oblique rotation up and to the right, the right superior oblique would help the right externus, but would hinder the right superior rectus. The inferior oblique of the left eye would hinder the left internus, but would help the left superior rectus. The right first basal center, therefore, must help to stimulate the right superior rectus, and the left third basal center must help stimulate the left internus. The basal center for the oblique muscles counteract the insufficiency of these muscles, whether the eyes be at rest or in motion.

Without the fusion centers, duction would be impossible; therefore, they can very properly be called duction centers. Each of the right and left first basal centers would produce superduction. Each of the right and left second basal centers would produce subduction. The right and left third basal centers would produce adduction. The right and left fourth basal centers would produce abduction. The right and left sixth basal centers produce minus cycloduction and the right and left seventh basal centers produce plus cycloduction.

The conjugate centers are verting and converging centers, and are not the centers that stand guard when oblique rotations and heterophoric conditions would cause the visual axes and the horizontal retinal meridians to leave the plane of the primary isogonal circle.

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## MEMORANDA

# TUBERCULIN AS A DIAGNOSTIC AND THERAPEUTIC AGENT IN THE TREATMENT OF CONJUNCTIVITIS ECZEMATOSA (PHLYCTENULAR CONJUNCTIVITIS AND KERATITIS)

## BASED ON THE STUDY OF FIFTY CASES *

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Phlyctenular conjunctivitis and phlyctenular keratitis (conjunctivitis eczematosis, phlyctenulosis) is one of the most frequent ocular maladies of childhood. The dominant characteristics of the disease are, first, its chronicity and, second, its tendency to recurrences. Usually it is not reckoned as a serious ocular malady, either in its clinical course or in its sequelæ. An extended experience, however, and a careful analysis of the clinical course and sequelæ of phlyctenular trouble neither justify this assumption nor satisfy the conscientious scientist with the results obtained by present methods of treatment. When there is taken into account the suffering experienced by many such patients, the prolonged invalidism they must undergo, the unheralded acute attacks of frequent occurrence which they much accept, the inroads on their visual acuity to which each new attack subjects them, the anxieties of parents as to the outcome of the attacks, the educational loss incident to the patient

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being compelled to remain out of school, the economic loss both to the child, to the parents and to the state, the physical retardation and interference with physical and mental development—when all these factors, which are not the unusual, but on the contrary, the common concomitants of a phlyctenular affection, are weighed and measured, the consideration of this disease then assumes a more serious aspect than a mere superficial consideration would lead one to expect. The full force of the hardships, incident to an attack or recurrence of the disease, is better appreciated when one considers that even the mildest attacks require from eight to fourteen days for complete recovery, while in those not unusual cases, where outbreaks rapidly follow each other with slight intermission, the period of invalidism may be prolonged into months or even years. The present recognized treatment of an acute attack of phlyctenular trouble, while far from satisfactory, might fairly be said to be ideal in results, compared to the extremely unsatisfactory results usually obtained by present-day methods in preventing recurrences of the disease. The prevention of these recurrences is the crux of the question, the pressing, insistent problem which confronts the clinician. Each recurrence subjects the patient to additional hazard in the possible limitation of his vision, besides producing and accentuating the manifold ill effects previously alluded to. The largest opportunity and the broadest possibility lie in the prevention of the recurrences. When this phase of the management of such cases has been definitely overcome, the application of the new therapeutic management will necessarily prove equally satisfactory in managing acute attacks of the disease. While the etiology has not been specifically established, phlyctenular disease has, by common consent, been considered to be due to a strumous constitution, a scrofulous diathesis, which, translated into modern scientific language, is interpreted as meaning an active or latent tuberculosis. The attacks were observed in connection with some lowering of the general body resistance and a minus resistance of the patient; almost always in association with the "adenoid syndrome"; frequently encountered with tuberculosis of bones and joints and common accompaniments or sequelæ of the exanthemata, measles, scarlatina, whooping-cough, or some debilitating illness. In the past scrofula or what was termed a "scrofulous diathesis," was held largely accountable for the production of the disease. Modern observers have established what might be termed the "pathologic kinship" existing between scrofula and tuberculosis. This kinship, while perhaps not positively established, has been so well recognized that the Germans speak of those exhibiting the scrofulous diathesis as "candidates for tuberculosis." Investigators have directed their attention especially to tuberculosis as an etiologic factor in the production of the disease.

Mr. Stephenson[1] states: "While nobody asserts that the phlyctenule itself is of tuberculous histologic structure, or that it contains tubercle bacilli, nevertheless it is most ordinarily believed that practically speaking the characteristic lesion occurs only in those who are subjects of tuberculosis, latent or otherwise."

Since Koch's discovery of the specific pathogenic microorganism of tuberculosis and his advocacy of tuberculin as a diagnostic and therapeutic agent, increased impetus has been given the investigation of the subject. The diagnostic measures now available, together with the improved technic for the recognition of tuberculosis, as well as the enlightened public opinion on the subject of tuberculosis, which insures a cooperation on the part of patients hitherto unknown, has been of incalculable advantage in conducting such an investigation.

The announcement of the discovery of tuberculin by Robert Koch created a furore among scientists and laymen. For a considerable period, notwithstanding the earnest protest of its discoverer, it was hailed as the long-looked-for specific in the treatment of tuberculosis. Disappointment in the results obtained by its indiscriminate use soon gave place to ridicule, and ridicule to abuse, and soon both the discoverer and his discovery were enveloped in a maelstrom of unseemly criticism. A small but earnest band of scientific workers, who were neither carried heavenward by the gale of enthusiasm following Koch's announcement, nor precipitated to the bottom of the scientific sea by the wave of criticism which superseded the first enthusiasm, continued to carry on their observations with the new agent. To the dignified insistent attitude of the great master, Koch, during these trying periods, and to the work of his faithful following, suffering humanity and the scientific world is indebted for a revival of the use of tuberculin along the lines first advocated by the great discoverer. "Koch's intention[2] was to create antitoxins by carefully introducing tubercle toxin into the body of the patient and through introduction of dead, specifically prepared tubercle bacilli in the newer preparation, to produce defensive substances against the morbid agent itself, to 'actively immunize' the patient, as the bacteriologists term it." While probably the therapeutic successes of tuberculin might at this time provoke an animated discussion, the same unsettled opinion does not obtain concerning its diagnostic efficacy. No less an authority than Wolff-Eisner[3] uses the following emphatic language in discussing its diagnostic value: "Contrary to what has been said concerning its therapeutic value, its diagnostic importance is not to be doubted, and it is hardly an exaggeration to maintain that this discovery stands at the beginning of the diagnostic science of tuberculosis, and our clinical knowledge concerning the distribution of this disease. It seems to have been the mission of tuberculin completely to fill up the gap caused by the loss of the value of the tubercle bacilli for purposes of early diagnosis, and to constitute an extremely fine biologic method by which to discover even the very first stages of tuberculosis."

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[1] Stephenson: Brit. Med. Jour., April 16, 1910.

The various tuberculin tests for the specific diagnosis of tuberculosis are four in number:

1. The cutaneous (von Pirquet).
2. The percutaneous (Moro, Doganoff, Lignières).
3. The conjunctival (Calmette, Wolff-Eisner).
4. The subcutaneous (hypodermic).

An extended consideration of the relative value of these four tests, with their modifications, is not within the province of this paper. In the selection of a test suitable for the observations which formed the basis of this report the limitations of and adaptability to the clinical material utilized became a matter of major importance. The hypodermic (subcutaneous) test was not applicable, principally because of the fact that with this ambulatory clinical material it was found impossible to carry out with precision the important details, such as keeping the temperature records, etc., which this test requires. The conjunctival test (Calmette, Wolff-Eisner) was not applicable because it is contra-indicated in any inflammation of the eye or lid; other hazards, such as a prolonged conjunctivitis and ulceration of the cornea, have also been reported from its use. The percutaneous test (Moro, Doganoff, Lignières) is said to be not so dependable as the von Pirquet test, principally because of the variable quality of absorption and the irritability of the skin differing in the different individuals. The cutaneous diagnostic tuberculin test of Pirquet was selected because it lends itself admirably to the investigations of the tuberculous ocular maladies of childhood. Von Pirquet states that the cutaneous test which bears his name is of value chiefly in children. In addition to its special application to suspicious tuberculous ocular diseases of childhood the test commends itself, first, for its comparative freedom from dangers; second, its simplicity of application; third, its freedom from unpleasant and disturbing reactions, focal, constitutional and local; fourth, elimination of prolonged and frequent periods of observation for confirmation; fifth, its reliability of results. Von Pirquet[4] thus explains the origin of the test:

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[2] Junius: Ophthalmology, Seattle, 1910, vi, 200.

[3] Wolff-Eisner: The Ophthalmic and Cutaneous Diagnosis of Tuberculosis, p. 11.

> I had found that in vaccination against small-pox only those persons show local symptoms within twenty-four hours who have formerly been vaccinated. One may therefore conclude if any early reaction is obtained that the person has been previously vaccinated. In applying the method of vaccination to tuberculosis, I found that, in the same way, an early reaction at the point of vaccination with Koch's tuberculin proves a previous infection with tuberculosis, and that this cutaneous reaction corresponds to Koch's fever reaction after injection with tuberculin.

The value of the cutaneous reaction as a diagnostic measure has been definitely established not only by extensive clinical observation, but by the post-mortem findings of von Pirquet. Two hundred of his cases, which had previously undergone the cutaneous test, came to autopsy. In these 200 cases were 109 children with no tuberculosis. In eighty-nine cases tuberculosis was found post mortem, and of these eighty-nine cases, sixty-five had given a positive reaction. No reaction had followed in twenty-four of the eighty-nine. Von Pirquet explained this negative result in the twenty-four, by the statement that no reaction occurs in the terminal stage of the disease, in the last stages of meningitis, in generalized tuberculosis, in miliary tuberculosis, in certain stages of measles and also that the reaction ceases as the child gets older. The twenty-four negative reactions were explained by their being classed in one or the other of these groups. All the children who had given a negative reaction were found on autopsy to be free from tubercle. Von Pirquet's conclusion[5] is that "the positive result of the reaction indicates with certainty the presence of active or inactive tuberculous changes." A negative result, when tuberculosis is present, is explained by von Pirquet on the premises previously alluded to. Adults react positively much more frequently than children, on account, it is alleged, of the greater frequency of tuberculosis in the adult. The test is, therefore, more useful in early childhood. Von Pirquet states that a negative result in the adult, after three attempts, proves with certainty that the patient is free from tuberculosis.

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[4] Von Pirquet: Cleveland Med. Jour., vii, No. 11.

In the series which I studied, a careful record was kept of the cases and every essential detail thought to be necessary was carried out faithfully and energetically. The period of observation approximates six months, and the cases studied numbered fifty. A systematic plan of observation was adopted and included:

1. Age of the patient.
2. Type of disease.
3. Duration of disease, with particular reference to frequency of "recurrences," and association with other diseases.
4. Family history, with particular reference to tuberculosis and syphilis.
5. Personal history, with particular reference to tuberculosis and syphilis.
6. General condition of patient.
7. Lymphoid complications, adenoids, enlarged tonsils, adenitis, etc.
8. Results of von Pirquet tests.
9. The local, focal and constitutional effects of the test.
10. Therapeutic administration of tuberculin.
11. The local, focal and constitutional effects of the therapeutic injection.
12. Results of the therapeutic injection on the ocular disease.

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[5] Von Pirquet: Wien. klin. Wchnschr., 1907, No. 38.

AR DISEASE

| Case No. | Age of Patient, Years | Type Ocul Dise | Temp. with V. Pirquet | Therapeutic Duration of Treatment‡ | Temp. Following First Therapeutic Injection | Result on Ocular Disease of Injection |
|---|---|---|---|---|---|---|
| 1 | 10 | Phlyct. Co | Negative... | March 15.... | 100 | Cured |
| 2 | 9 | Phlyct. Ke | Negative... | ............ | ..... | Cured |
| 3 | 3 | Phlyct. Co | Negative... | March 20.... | 99.6 | Improved |
| 4 | 3 | Phlyct. Ke | Negative... | February 27.. | 99 | Cured |
| 5 | 12 | Double Ph<br>Kerat. C | Negative... | January 25... | 100.2 | Marked improvement |
| 6 | 1 | Phlyct. Ke | Negative... | ............ | ..... | Improved |
| 7 | 18 | Phlyct. Ke<br>Epi. Sc | Negative... | March 1..... | 99.4 | Improved |
| 8 | 11 | Phlyct. C | 100....... | No treatment. | ..... | Unknown |
| 9 | 42 | Keratitis | Negative... | March 1..... | 99 | Improved |
| 10 | 4 | Phlyct. K | Negative... | March 15.... | 100 | Improved |
| 11 | 22 | Phlyct. C | Negative... | February 27.. | Negative | Cured |
| 12 | 6 | Phlyct. K | Negative... | January .... | 99 | Cured |
| 13 | 9 | Phlyct. Ke | 99 (after third test). | ............ | ..... | Improved |
| 14 | 7 | Phlyct. K | Negative... | February 6... | 99.2 | Cured |
| 15 | 8 | Phlyct. K | Negative... | February 27.. | 100.2 | Improved |
| 16 | 3 | Phlyct. K | Negative... | February 27.. | 99.2 | Cured |
| 17 | 13 | Phlyct. K | Negative... | October ..... | 99 | Improved |
| 18 | 56 | Phlyct. K | Negative... | October ..... | 99.4 | Cured |
| 19 | 6 | Phlyct. K | Negative... | October ..... | 99 | Cured |
| 20 | 2 | Phlyct. K | Negative... | ............ | ..... | Cured |
| 21 | 4 | Phlyct. K | Negative... | January .... | 99.4 | Cured |
| 22 | 10 | Phlyct. C | Negative... | February 27.. | 99 | Cured |
| 23 | 26 | Phlyct. C | Negative... | February 20.. | 99.3 | Cured |
| | 9 | Phlyct. C | Negative | March 20 | 99 | Cured |

the papule and hyperemic zone varies, though neither the size nor the intensity is held to be a measure of the

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5. Von Pirquet: Wien. klin. Wchnschr., 1907, No. 38.

In addition to the therapeutic use of tuberculin the patients were given the usual local ocular treatment of conjunctivitis eczematosa (boric solution, yellow oxid of mercury, calomel, atropin, warm compresses, etc., as indicated) and constitutional measures such as regulation of diet, hygienic conditions, systematic life, exercise, fresh air, tonics, etc., and where complications, such as hypertrophied tonsils, nasal obstructions, adenoids or other diseased conditions existed, these were corrected or treated. The von Pirquet cutaneous reaction test was first applied in all cases, and in the event of a positive reaction ensuing, the therapeutic injections were instituted, after the local reaction and all other untoward developments had subsided completely.

Three negative tests were elicited before the patient was considered free from suspicion of tuberculosis. The technic of the von Pirquet test was as follows: The inner side of the forearm, at the point selected, was cleansed with ether, and two drops of Koch's old tuberculin, undiluted, were placed on the skin about four inches apart. The special scarifier, previously sterilized, which is supplied with the tuberculin, was applied vertically first to a point midway between the drops and with a circular movement; a very superficial, bloodless scarification was accomplished—this constituted the "control." Next, the scarifier was applied to the first drop and a similar scarification performed; and finally it was applied to the other drop.

A drop of 1 per cent. phenol in glycerin was placed in the "control." A light sterile dressing, secured by adhesive strips was applied, and the patient directed to report within forty-eight hours for inspection.

Following Wolff-Eisner's suggestion the local reactions were divided into three degrees:

1. Distinct reaction.
2. Strong reaction.
3. Very strong reaction.

These terms are self-explanatory, and after limited observation no difficulty is encountered in assigning the reaction to its proper class or degree. A reaction was characterized as positive when, within forty-eight hours the appearance at the site of the inoculation exhibited a papule surrounded by a hyperemic zone. The size of the papule and hyperemic zone varies, though neither the size nor the intensity is held to be a measure of the extent of the tuberculous process. The reaction of the "control" is, as a rule, very slight and easily differentiated from the inoculation scarifications. The behavior of the reaction, the time of its appearance, the duration of its existence, its intensity and size have each been the subject of much speculation. At the present time, however, no sufficiently positive corroborative conclusions are available covering the theories advanced. The Tuberculin Bacillen Emulsion (B.E.) human type, serial dilutions (Mulford) was used for the therapeutic injections. This product is marketed in six vials, known as Serial Dilutions No. 1 to No. 6, inclusive. Each two minims of Dilution No. 1 contains 1/10,000 milligram; each two minims of the succeeding dilutions is ten times stronger than the preceding solution; for example, two minims of Dilution No. 2 contains 1/1,000 milligram; two minims of Dilution No. 3 contains 1/100 milligram.

The proper administration of tuberculin is emphasized by all observers of large experience. Koch[6] advises that "the best way of guarding against the misapplication of tuberculin is to use it in cases in which the temperature of the body does not exceed 37 C. (98.6 F.)." Apropos of the faults of administration, Trudeau[7] writes as follows:

> I quite agree with Denys that the principal faults in treatment—faults which may lead to failure and even seriously endanger the patient's chances—are: beginning the treatment with too large amounts; raising the dose too rapidly, or at too short intervals; injecting again before all effects of the reaction, both constitutional and local have passed away; increasing the dose after a reaction has occurred; neglecting to consider malaise, headache, loss of appetite, and increased cough, as evidence that the limit of the patient's tolerance has been reached and calls for an interval of rest and a reduction of the dose. The physician who disregards, as of no importance, an increase of a minute fraction of a milligram of tuberculin or a rise of a few tenths of a degree of temperature will meet with disappointment and disaster in the use of tuberculin treatment.

In the question of administration the main factor is the dosage. Each case must be individualized, there being *no average dose;* 1/10,000 milligram, for instance, might be too large a dose for one individual, and one milligram too small a dose for another. In harmony with these observations I followed the plan of beginning, in most cases, with an injection of 2 minims of Bacillen Emulsion, Dilution 1, 1/10,000 milligram; in infants and young children only 1 minim of Dilution 1 was used. If no elevation of temperature occurred about twenty-four hours afterward, or other evidences of reaction, such as marked reaction at the site of injection, nausea, headache, general malaise, etc., appeared, the injection was repeated, usually twice a week, each dose being increased by 2 minims until 20 minims of that dilution was injected; after which Dilution 2 was begun and proceeded with in the same manner. When 20 minims of Dilution 2 was reached, Dilution 3 was taken up and so on, in like manner, the entire series of dilutions were, if possible, exhausted. Any evidence of intolerance, which was exhibited by the development of the symptoms above described, was a signal for reducing the dose, omitting it entirely, or extending the interval of injection; in any event the conduct of the administration was governed entirely by the reaction or toleration exhibited by the patient.

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[6] Koch: Pulmonary Tuberculosis, Philadelphia, 1907.
[7] Trudeau: Am. Jour. Med. Sc., cxxxii, 175.

The majority of observers agree that haste in the administration is responsible for much disappointment in the use of tuberculin, and most of them suggest carrying the patient along slowly and cautiously, over a period of at least six months and better still, of one year.

## ANALYSIS OF CASES STUDIED

An analysis of the cases studied, shown in the accompanying table, discloses the following facts:

1. *Age of the Patients.*—The youngest patient was 1 year old; the oldest, 56 years; the average age 12¼ years, approximately.

2. *Type of Ocular Disease.*—There were twenty-four cases of phlyctenular conjunctivitis (48 per cent.). The right eye was involved in eleven of the twenty-four cases (45.8 per cent.). The left eye was involved in five of the twenty-four cases (20.8 per cent.). Both eyes were involved in eight of the twenty-four cases (33⅓ per cent.). There were twenty-six cases of phlyctenular keratitis (52 per cent.). The right eye was involved in seventeen of the twenty-six cases (65.3 per cent.). The left eye was involved in three of the twenty-six cases (11.5 per cent.). Both eyes were involved in six of the twenty-six cases (23 per cent.).

3. *Duration of Disease.*—The shortest period, since first attack, was two days. The longest period, since first attack, was five years.

4. *Lymphoid Complications.*—In thirty-two of the cases enlarged or diseased tonsils, adenoids and cervical adenitis were found coexisting (64 per cent.). In nine of the cases cervical adenitis was found alone (18 per cent.). In eight of the cases enlarged or diseased tonsils and cervical adenitis were found (16 per cent.). In one of the cases, the tonsils and cervical glands had been removed.

5. *Tuberculous Family History, Syphilis, etc.*—In ten of the cases a positive family history of tuberculosis was obtained (20 per cent.). In none of the cases was a family history suspicious of syphilis elicited.

6. *Personal History of Active or Latent Tuberculosis, Syphilis, etc.*—In six of the cases a personal history of active or latent tuberculosis was obtained (12 per cent.). In none of the cases was a personal history or clinical evidences of syphilis obtainable.

7. *Von Pirquet Cutaneous Diagnostic Test.*—In forty-six cases a positive cutaneous reaction to the test was obtained (92 per cent.).

8. *Ocular Reaction Accompanying the von Pirquet Test.*—In four cases a varying reaction was obtained following the diagnostic test (8 per cent.).

9. *Temperature Accompanying the von Pirquet Diagnostic Test.*—In five cases an elevation of temperature following the test was observed (10 per cent.).

10. *Therapeutic Duration of Treatment.*—The longest period of treatment was six months; the shortest period about three weeks.

11. *Temperature Following the First Therapeutic Injection.*—In forty-four cases there was a rise in temperature following the first therapeutic injection (88 per cent.). The highest elevation recorded was 100.2 F.; the lowest elevation recorded 99 F.

12. *Result on Ocular Disease of Therapeutic Injection.*—In thirty-two cases the ocular disease had cleared up completely and there had been no further recurrences up to date; these patients were tabulated as "cured" (64 per cent.). In thirteen cases the ocular disease had "improved" but still required treatment (26 per cent.). In two cases the ocular disease had cleared up completely and the diagnostic test proving negative, no therapeutic injections were used (4 per cent.). In two cases the ocular disease had "improved" but still required treatment; a negative von Pirquet being obtained no therapeutic injection was used (4 per cent.). In one case patient did not return for treatment.

In the study of the foregoing cases two main points were to be determined; first, the etiologic relationship existing between tuberculosis and conjunctivitis eczematosa; second, the value of tuberculin as a diagnostic and therapeutic agent in the treatment of conjunctivitis eczematosa. After a careful consideration, I am convinced that conjunctivitis eczematosa should be regarded as a manifestation of constitutional tuberculosis. It is impossible longer to ignore the evidences which point unmistakably to the scientific correctness of this view. A careful study of the symptom-complex this class of cases exhibit, with every stigma of a tuberculous process accentuated and occurring with startling constancy in each individual case, and these evidences supported and corroborated in unmistakable manner by the investigations of numerous observers leads to but one positive conclusion, and that is that conjunctivitis eczematosa is an ocular manifestation of a constitutional tuberculosis, and that tuberculin is the appropriate and especial agent for its proper treatment.

The clinical course of the malady, with its chronicity and tendency to recurrences; its predilection for the poorly nourished and those with a minus resistance; its glandular complications; its response to the tuberculin diagnostic and therapeutic measures all confirm and emphasize this belief and forces on us a definite line of therapeusis—the proper administration of tuberculin. The preponderance of the rapidly accumulating mass of evidence collected by numerous observers adds weight and undoubtedly will persist in substantiating the correctness of this conclusion. I am convinced from a careful survey of the investigations of others as well as from a study of my own cases that conjunctivitis eczematosa is an ocular manifestation of constitutional tuberculosis and that its proper management must include not only the usual ocular or local treatment, but more particularly the general or constitutional measures appropriate to the treatment of tuberculosis. At the head of the constitutional agents advised in the treatment of tuberculosis I would unhesitatingly place tuberculin. The favorable results experienced from its use by a large number of observers cannot be denied; the theoretical correctness of the remedy is undoubted and the increasing favor its discriminate and painstaking employment evokes recommends it as a remedial agent of especial value and dependability in the treatment of diseases of tubercular origin.

## CONCLUSIONS

The results obtained from the study of the cases forming the basis of this report justify the following conclusions:

1. The tuberculous nature of conjunctivitis eczematosa is established and this opinion is supported by the results obtained in the cases subjected to tuberculin, both diagnostically and therapeutically, and by the majority of the other essential clinical findings.
2. The cutaneous diagnostic tuberculin test of von Pirquet is a dependable method for the recognition of tuberculosis, particularly in children.
3. Tuberculin as a therapeutic agent in the treatment of conjunctivitis eczematosa is an agent of distinct and special value and should be employed faithfully, judiciously and perseveringly in this class of cases.

**I wish to express my indebtedness to Dr. J. B. Murphy for suggesting to me the use of tuberculin in this class of cases; to Dr. H. Woodruff, surgeon to the Illinois Charitable Eye and Ear Infirmary, and chief of our clinic, for permission to conduct the major part of these observations; also to Doctors Schenck, Hoffman and Smith, of the House Staff of the Infirmary for valuable assistance in carrying on the observations.**

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## MEMORANDA

## MEMORANDA

# DISTORTIONS OF THE VISUAL FIELDS IN CASES OF BRAIN TUMOR

## (*Second Paper*)

### DYSCHROMATOPSIA IN RELATION TO STAGES OF CHOKED DISK *

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In a former paper,[1] introductory to this series, it was related that reliable perimetric observations had been possible in 123 patients in a series of 200 cases of cerebral tumors. These had shown normal fields in twenty-seven instances, a tendency toward hemianopsia in forty-two, and color interlacing or inversion (dyschromatopsia), with more or less symmetrical constriction, in fifty-three instances. It is our present purpose to give specific illustrations of the cases showing dyschromatopsia and to correlate them, so far as possible, with the stages of an advancing choked disk, especial emphasis being laid on the field deviations coincident with, or antecedent to, the earliest ophthalmoscopic changes.

There have been ten cases of tumor which have shown color-interlacing, either in the total absence of a choked disk or with a very incipient process observable in the eye-grounds. It is a natural conclusion, therefore, that these represent cases in which definite localizing (irritative) symptoms have led to a precocious surgical intervention and consequently to the best operative results. Thus in these ten cases the tumor was enucleated in five instances, was exposed and considered irremovable in one, and was unlocalizable and not disclosed by the operation (decompression) in four. In all of them the normal color relations were restored after the operation.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Los Angeles Session, June 27-30, 1911. Publication rights reserved by the American Medical Association.

[1] The first paper in this series (The Johns Hopkins Hospital Bulletin, June, 1911) gave some statistical studies of the results of perimetry in our series of 200 cases of brain tumor. The succeeding three papers will be devoted, respectively, to the binasal, bitemporal and homonymous hemianopsias observed in these cases.

It is possible for convenience in this analysis to subdivide these cases which have exhibited early color distortion into three groups: (1) those in which, in the total absence of even an incipient grade of choked disk, interlacing was bilaterally equal; (2) those in which definite interlacing was unilaterally represented; (3) those in which interlacing coincided with a low grade of choked disk, but disappeared after a decompression, before the subsidence of the edema. Illustrations may be given of each of these groups.

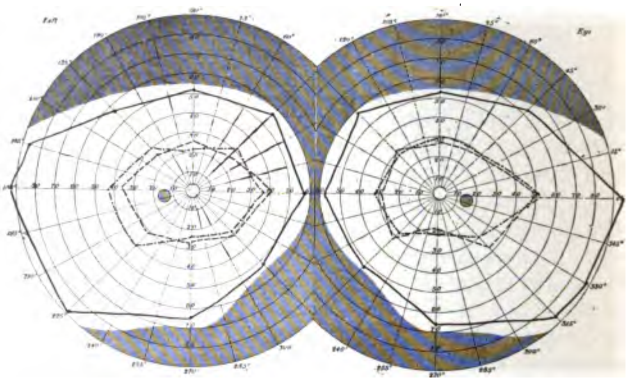

Fig. 1.—Charts of Case 1 before operation, showing characteristic blue and red interlacing with some constriction of color-fields.

### GROUP I. DYSCHROMATOPSIA ANTECEDENT TO ANY OBSERVABLE CHANGE IN THE NERVE-HEAD

The following case shows that an enormous growth, if slowly advancing and unaccompanied by coincident cerebral edema, may progress for years without producing any of the usual pressure manifestations whatsoever, and that distortion of the color-fields may be the earliest indication of their appearance.

Case 1.—Surgical No. 25,396. *Enormous endothelioma of right hemisphere causing focal epilepsy and hemiparesis but no pressure symptoms except a slight grade of color interlacing.*

*History.*—Dr. W., aged 49, was admitted Feb. 4, 1910. The patient received a blow on the cranial vertex in 1898, and six years later began having attacks of focal epilepsy, starting with an aura in the left foot. There was gradual loss of con-

**trol of the left foot and hand, with marked spasticity. An examination in April, 1909, disclosed no pressure symptoms whatsover, aside from the fact that though the color-fields were wide there was some slight interlacing of the blue and red outlines. During the succeeding ten months the patient, who was leading an active life, was repeatedly observed, but there was no apparent advance in the symptoms. He had never had a headache; never any nausea; never diplopia. There was some fulness of the retinal veins but no edema. Visual acuity was normal. The fields on Feb. 4, 1910, showed practically the same conditions as on the examination ten months before (Fig. 1).[2]**

**_Operation._—On February 5 and February 9, in two stages, an endothelioma weighing 198 gm.—the largest tumor in our series successfully extirpated—was removed from the right hemisphere. In view of its large size one would have expected at least some pressure encroachment on the upper fibers of the radiation which would have produced a "slant" at least of the contralateral (left) fields in their lower portion.**

**February 25, the fields showed a complete restoration of the normal outlines. August 26 (six months later), the normal relations persisted (Fig. 2).**

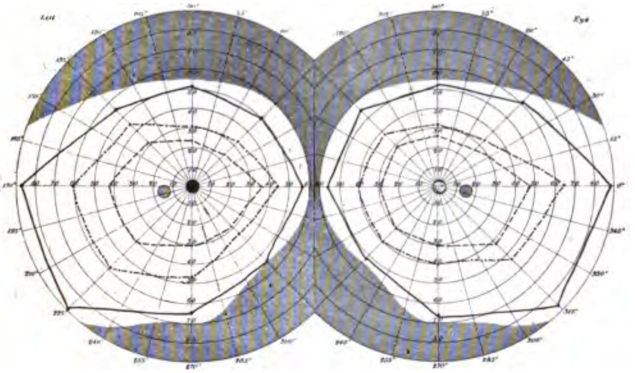

**Fig. 2.—Charts of Case 1 after operation, showing restoration of normal color relations after tumor removal.**

There are two other cases in the series in which Jacksonian attacks represented the chief clinical manifestations of the lesion. Both came under observation at an early stage, before the occurrence of any recognizable changes in the eye-grounds. The distortion of the fields

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**[2] In all of these charts the usual dot and dash indicates the blue; the broken dash, the red periphery.**

was more marked, if anything, on the side contralateral to the lesion in both cases.

Case 2.—Surgical No. 18,602. *Glioma originating in left motor cortex, causing focal epilepsy with color-interlacing as the only evidence of pressure.*

*History.*—Miss T. M., aged 22, was admitted Dec. 31, 1905. Attacks of focal epilepsy with an aura in the right foot, succeeded by weakness of the leg, followed a cranial injury received six months before. There were no pressure-symptoms, except the very definite interlacing (at that time not recognized as an evidence of pressure). The ophthalmologic condition was normal in all other respects. No trace of neuroretinal congestion or swelling. On January 10 a glioma the size of a pigeon's egg was removed from the mesial end of the left precentral convolution.

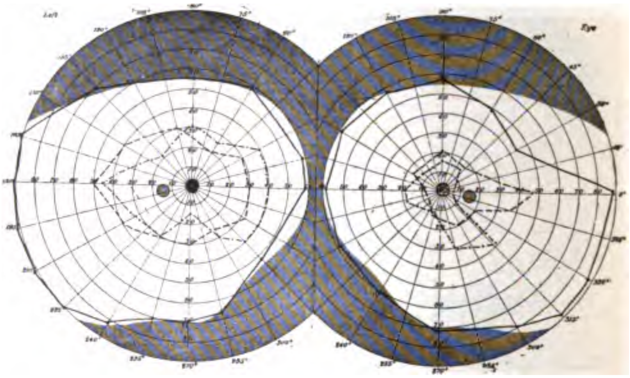

Fig. 3.—Charts of Case 2 before operation. Color-interlacing and constriction more marked on the right.

It is to be noted that the right field (Fig. 3) shows some tendency toward an upper temporal defect which may indicate a beginning implication of the left visual pathway, though the defect is not bilaterally represented. An extradural clot formed in this case, and on the day after the operation the patient gradually became stuporous and a definite choked disk was then found. The flap was reelevated and the clot removed, but she did not survive. There are no postoperative fields, therefore.

Case 3.—Surgical No. 23,150. *Syphiloma of right inferior parietal lobule causing focal epilepsy with sensory aura in left hand. Color-interlacing without choked disk.*

*History.*—Mr. A. S. J., aged 47, was admitted Nov. 3, 1908. The patient had had attacks of focal epilepsy beginning with a sensory aura in the left hand for the previous six months, with some right-sided headache.

*Examination of Eyes.*—Pupils unequal; right 7 mm., left 4 mm.; irregular contour. This condition was noted a year before present symptoms were observed. Right pupil reacted promptly to light; left sluggishly. Consensual reaction poor from right to left; fundi negative; acuity normal. Fields (Fig. 4) showed typical interlacing, with superior constriction (due to deep-set eyes and overhanging brows), particularly of the left form field, and an almost total inversion of blue within red on the right. It is interesting that the patient is a buyer for a large drygoods establishment and is considered an authority on colors.

*Operation.*—November 7. A large fibrous tumor (histologically a syphiloma) was disclosed and removed from the right inferior parietal region. There were no postoperative complications. Inequality of the pupils had practically disappeared by November 10, though the right remained somewhat larger than the left.

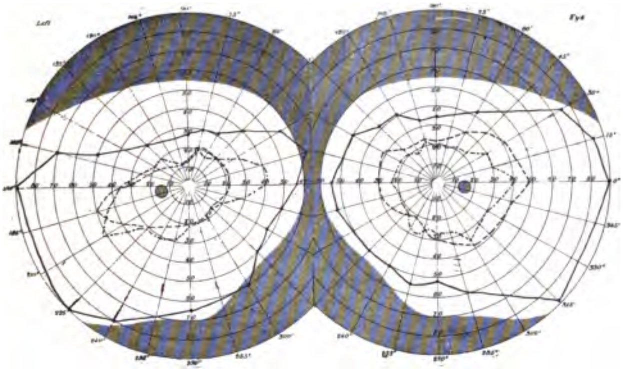

Fig. 4.—Charts of Case 3 before operation, showing interlacing, with almost complete inversion for the right eye. Superior flattening of the fields due to overhanging brows.

November 29 the color-fields were found to have resumed their normal relations without interlacing, though a slight irregular constriction persisted.

## GROUP II. UNILATERAL DYSCHROMATOPSIA ACCOMPANIED BY AN INCIPIENT GRADE OF CHOKED DISK

A number of examples of unilateral dyschromatopsia accompanied by an incipient grade of choked disk occur in the series, both conditions being more marked on the side of the lesion. Two illustrations will suffice. In the first case it will be seen that the interlacing was much more definite on the side of the lesion. In the second case the degree of interlacing was about equal on the two sides, but there was more constriction on the side of the lesion. These and other cases suggest that the degree of interlacing may possibly prove to be of some localizing value, so far as the hemisphere involved is concerned, in very much the same way as the relative degree of choking in the two eyes may sometimes prove of value in determining the side of the lesion.

Case 4.—Surgical No. 24,408. *Gliomatous "hypertrophy" of median right precentral convolution with focal epilepsy, headaches and color-interlacing, more marked on the side of the lesion. Incipient unilateral choked disk.*

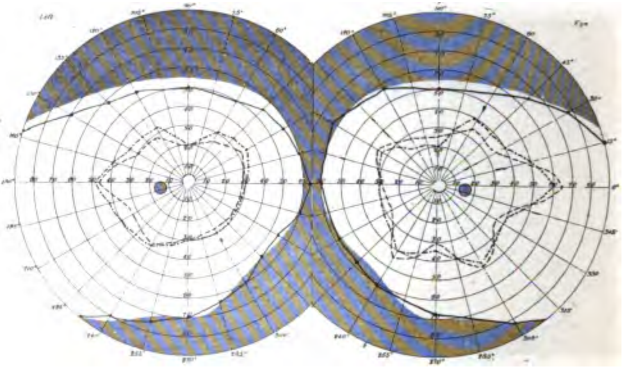

Fig. 5.—Charts of Case 4 before operation, showing interlacing confined to the right field, the side of the lesion.

*History.*—Mrs. W. J., aged 33, was admitted July 12, 1909. Focal epilepsy with motor aura in left hand, of five years' duration; attacks more frequent and more severe of late, usually ending in a general convulsion; some headaches and occasional nausea; some slight subjective blurring of vision; progressive weakness of left hand and arm.

*Ophthalmologic Examination.*—Pupils equal; reactions normal; no oculomotor disturbances. Fundi: Some fulness and tortuosity of the veins and slight haziness of nasal margin on right; no measurable swelling; left fundus normal. The fields (Fig. 5) show characteristic blue and red interlacing, limited to the right eye, though the left shows a tendency in this direction. Acuity 20/20, both eyes.

*Operation.*—July 14. Extirpation of a large, firm and sharply outlined gliomatous tumor limited to the central portion of the pre-Rolandic gyrus; flap replaced without drainage; no subsequent complications. There was a slight postoperative edema of the disks in both eyes, with considerable haziness of the nasal borders.

The fields August 9, the day of the patient's discharge, showed a restoration of normal color relations, though there was still some constriction and irregularity of outline.

June 18, 1910, the patient returned for examination. She was well. The eyes were absolutely negative, with unusually wide color-fields (Fig. 6), with regular outlines and normal relations. Acuity: Right, 20/20; left, 30/20.

Case 5.—Surgical No. 27,397. *Tumor (uncertified) of left hemisphere, producing focal epilepsy. Homolateral color-interlacing and choked disk, disappearing after decompression.*

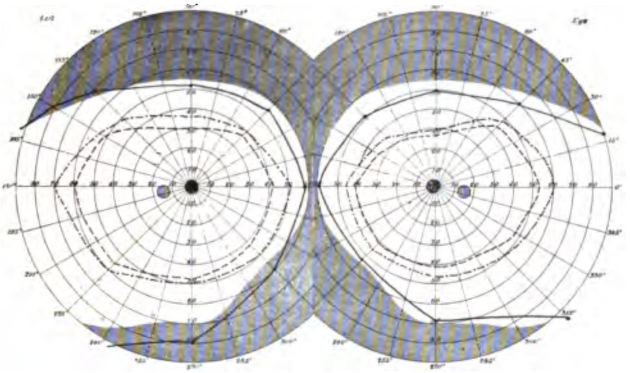

Fig. 6.—Charts of Case 4 ten months after tumor removal, showing unusually wide and non-interlaced color-fields.

*History.*—Mrs. H. J., aged 49, was admitted Feb. 27, 1911. Attacks of focal epilepsy in right arm for several years, becoming severe, with occasional general convulsions. Some weakness of hand and foot of late, and thickness of speech. Headache, referred to left parietal region; never severe.

*Examination of Eyes.*—Pupils equal; reactions normal. Oculomotor: Some nystagmoid twitches both to right and left. Fundi: Right, practically normal with exception of some fulness and tortuosity of veins; left, obscuration of nasal margin and physiologic cup by edema; no measurable swelling; veins full and tortuous. Acuity: Right, 15/15; left, 15/25. Fields (Fig. 7) showed some constriction with definite interlacing on the left and irregularity of color outlines on right (presaging interlacing?).

February 28. Osteoplastic exploration revealed increased tension; no cortical lesion. Subtemporal decompression produced a complete subsidence of symptoms.

March 27. Examination revealed the following conditions: Fundi: Right, normal; left, slight haziness persisted on nasal margin and lamina cribrosa was still obscured. Acuity: Right, 15/15; left, 15/10. Fields (Fig. 8): Normal relation restored, though there was some constriction of all boundaries.

### GROUP III. DYSCHROMATOPSIA ACCOMPANYING EARLY STAGES OF CHOKED DISK. RESTORATION OF NORMAL COLOR OUTLINES BEFORE THE COMPLETE SUBSIDENCE OF THE EDEMA

These cases, of which there are several in the series, tend to show that the color-interlacing is a more delicate manifestation of pressure than an observable degree of neuroretinal stasis and edema. A single illustration will suffice.

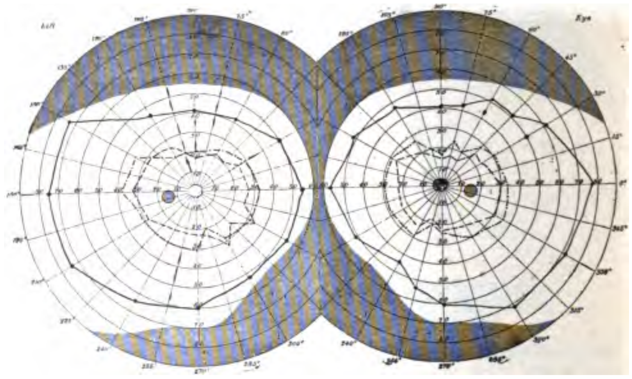

Fig. 7.—Charts of Case 5 before operation, showing interlacing confined to the left field, the side of the lesion.

Case 6.—Surgical No. 23,559. *Meningeal angioma of right temporal lobe. Recent headaches, color-interlacing and incipient choked disk the only symptoms of tumor.*

*History.*—H. R., colored, aged 39, was admitted Feb. 1, 1909. Transferred from Dr. Barker's service for decompression. Severe, constant temporal headaches for a few weeks. No localizing symptoms of tumor recognized.

*Examination of Eyes.*—Negative in all respects except for some haziness of disk margins, more evident on the left, where

**there was a measurable swelling of 1 D. The fields showed typical color-interlacing (Fig. 9).**

***Operation.*—February 3. Left subtemporal decompression disclosed an unsuspected angiomatous tumor of the temporal meninges. There was complete postoperative subsidence of all symptoms. On February 11 the measurable swelling had disappeared. On February 15 the fields (Fig. 10) had resumed their normal outlines. On March 2 the disks, though fairly clear, still showed some slight engorgement and hyperemia.**

During the past few years there has been an increasing tendency on the part of those interested in the pathogenesis of choked disk to regard the process, from its incipient condition of vascular injection to the terminal secondary optic atrophy, as merely stages of one and the same underlying pressure disturbance. The studies of Alfred Saenger, Sir Victor Horsley, Leslie Paton, George E. de Schweinitz, Eugen von Hippel, Franz Schieck and others, based largely on the investigation of cases after surgical relief from pressure, have contributed much toward the common acceptance of the views originally advanced in large part by Schmidt-Rimpler and Manz.

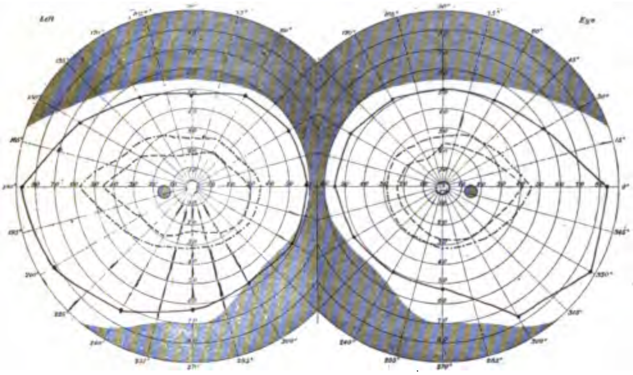

**Fig. 8.—Charts of Case 5 after decompression, showing restoration of normal color-relations. Some neuroretinal edema persisted in left eye.**

One particular source of confusion was the tendency of ophthalmologists to designate the early condition as an "optic neuritis" and to reserve the term "choked disk" for the later manifestations, when a measurable swelling of the nerve-head occurs.

Marcus Gunn, in his discussion of J. S. Risien Russell's paper,[3] did a service in subdividing the gradations of choked disk—called by him "tumor papillitis"—into stages. These stages are necessarily more or less arbitrary and possess no sharp lines of distinction, but they are useful in conveying to others, without a long description, some fairly definite idea of the severity of the condition which is being considered. They have been adopted with some changes, by de Schweinitz in his admirable paper read before this section two years ago.[4] We too have ventured to make some modifications, though the main subdivisions remain about as Marcus Gunn originally gave them. To the mere description of the ophthalmoscopic picture we have added a note on the perimetric deviations and the changes in visual acuity which may be expected in each stage. It is important to bear in mind that the changes in one eye are apt to be in advance of those in the other. This is particularly true of such supratentorial lesions as are unaccompanied by ventricular hydrops—cases, in other words, in which the tension in one hemisphere is greater than in the other.

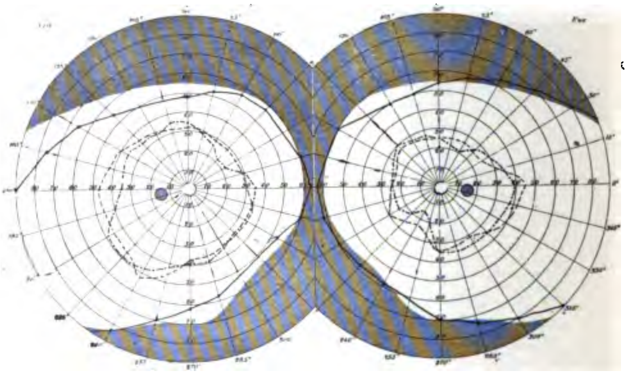

Fig. 9.—Charts of Case 6 in Gunn's Stage 1, showing bilateral interlacing as plotted before decompression.

*Stage 1.*—This shows some hyperemia of the disks with haziness of the upper and lower margins where they are crossed by the main vessels. This haziness gradually spreads over and obscures the nasal half of the disk, the temporal edge remaining clear. There may be some narrowing of the physiologic cup and the veins are slightly full and sinuous. Visual acuity is not affected. Color-interlacing occurs, and together with the retinal change may be more marked on the side of the tumor, if cerebral. A prompt restoration occurs after decompression.

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[3] Russell, J. S. R.: The Indications for Operation in Cases of Intracranial Tumor, Brit. Med. Jour., Oct. 26, 1907, p. 1120.

[4] De Schweinitz and Halloway: The Operative Treatment of Papilledema Dependent on Increased Intracranial Tension, Therap. Gaz., July 15, 1909.

*Stage 2.*—The swelling of the papilla, now obscuring the temporal as well as the nasal margin, has become measurable, the physiologic cup is filled in, and there may be an extension of the edema to the surrounding retina. There is an outspoken stasis with tortuosity of the veins. There is apt to be a subjective lowering of acuity. Interlacing is present with constriction. Rapid return to the normal follows relief of pressure.

*Stage 3.*—There is a further swelling of the papilla, which is now definitely prominent and spreads over a larger fundus area. Its margins are lost. Fine striations appear in the edematous retina, particularly between disk and macula. Retinal hemorrhages may occur and venous stasis is marked, the congestion showing itself in the dilated palpebral venules as well as in the eye-grounds. Measured acuity is usually below normal. There is constriction of all fields, often with color inversion, but normal relations are restored, and usually normal acuity, after adequate decompression.

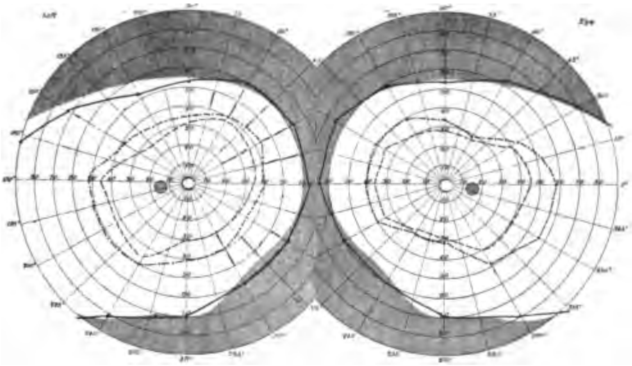

Fig. 10.—Charts of Case 6, showing return of normal relations two weeks after decompression.

*Stage 4.*—The papilla becomes more prominent and, losing its reddish color, appears more opaque. Hemorrhages may be more numerous and larger, and exudates similar to those seen in nephritis may be present on the disk or surrounding retina. Many cases, however, particularly those of slow onset, remain free from hemorrhages and exudates. There is new-tissue formation, giving a fluffy appearance to the disk. Acuity of vision is considerably lowered. There may be transient amaurotic periods. There is further central constriction and colors can often be recognized only in large sheets. Complete pressure relief, such as may be afforded by tumor extirpation, or by adequate decompression in a case with symptoms of short duration, may lead to an almost total restoration of vision, but if the condition at this stage has been of long standing and there is an excess of new tissue formation there may be a further postoperative lowering of vision as the edema subsides.

*Stage 5.*—There is a gradually decreasing vascularity of the papilla. Its surface becomes paler than normal and shows the fine, fluffy appearance due to new tissue formation, the contraction of which tends to produce a subsidence of the measurable swelling. This may disclose the shimmering atrophic disk as through a mist. The arteries tend to become of smaller caliber and the thickening of their perivascular lymph sheaths is apparent. This is the stage of atrophy with supposedly (Gunn) inevitable blindness. Acuity is practically reduced to the counting of fingers. There are frequent periods of temporary amaurosis. There is apt to be achromatopsia in one or both eyes, and small form fields (often requiring disks larger than 0.5 cm.) may be plotted. These often show a tendency toward a binasal hemianopsia with loss of central vision. Some vision may be preserved by operation.[5]

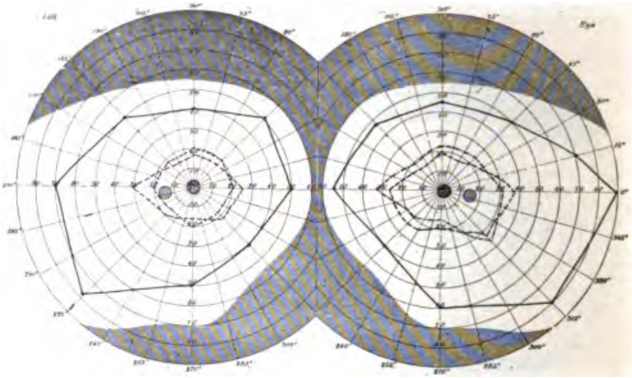

Fig. 11.—Charts of Case 7 before operation, showing interlacing and constriction accompanying Gunn's second stage of choked disk.

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[5] There are other subdivisions which might well be made, notably a Stage 6 descriptive of the fundus change which persists after total loss of vision in unoperated cases. It would be well also to incorporate with each stage a description of the tell-tale residual of the original process which follows complete postoperative subsidence of the edema.

This digression into the question of choked disk has been made for the purpose of allowing us to correlate the perimetric disturbances, more particularly the color-alterations, which are observable in these different stages. We have already seen that color-interlacing may actually precede the onset of the earliest stasis phenomena; also that in conditions of incipient though demonstrable edema of the nerve-head the color-interlacing disappears after a successful decompression before the ophthalmoscopic picture of the disk has entirely cleared. We have, in other words, already given records of perimetry not only for Gunn's first stage of choked disk, but also for certain antecedent states. Though these cases of early interlacing are the more important ones, it seems advisable to follow the perimetric changes through the succeeding stages.

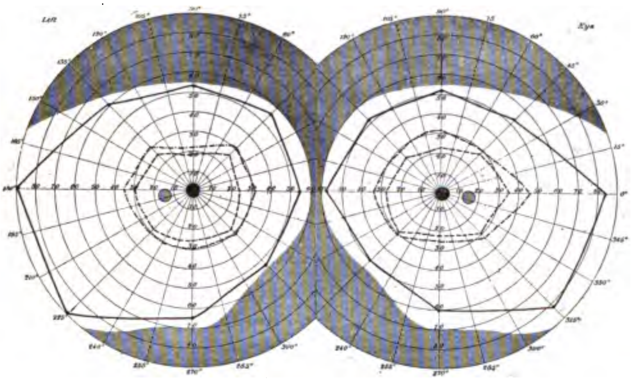

Fig. 12.—Charts of Case 7, showing postoperative restoration of normal color-relations before complete subsidence of second stage of choked disk. Some persisting constriction.

The following two cases have been chosen to illustrate Marcus Gunn's Stage 2, for they clearly show that the perimeter may be of almost greater value than the ophthalmoscope in determining the degree of severity of the process. The ophthalmoscopic picture in the first patient (Case 7) was clearly that of a low-grade, though long-standing, process, and yet the fields and measured acuity before and after operation show that the nerve-head had suffered permanent damage. In the second patient (Case 8) the condition of the fundi, with hemorrhages, a prominent papilla, marked perivascular streaking and some new tissue formation, would doubtless have been regarded as well along toward, or actually in, Stage 3, but the process subsided after the operation, leaving normal acuity, normal field outlines and no observable trace of its preexistence in the eye-grounds.

The case of long standing but low-grade pressure, illustrating the perimetric changes observed in Marcus Gunn's second stage of choked disk is as follows:

Case 7.—Surgical No. 25,339. *Endothelioma of left cerebellopontine angle. Choked disk. Interlacing of color-fields. Suboccipital exploration with removal of tumor.*

*History.*—Mr. A. J. B. was admitted Jan. 24, 1910. Symptoms of four years' duration were first noticed as an unsteadiness in gait, following an injury to the head. About a year before admission double vision occurred, and has persisted. For the previous six months there had been increasing deafness in the left ear, subjective tinnitus, vertigo, and, of late, considerable headache.

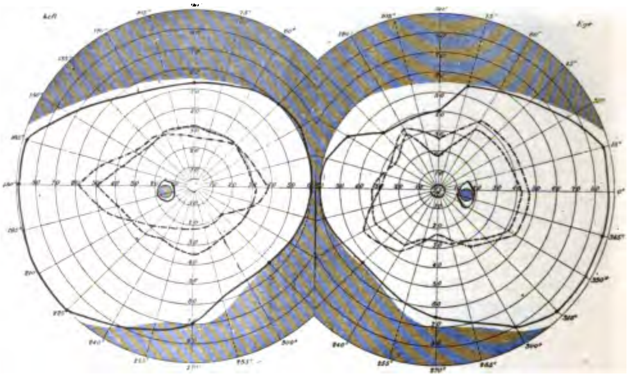

Fig. 13.—Charts of Case 8 before operation, showing interlacing without constriction accompanying a condition of the papilla approaching Gunn's Stage 3.

*Examination of Eyes.*—Pupils equal with normal reactions. Ocular movements normal, though convergence was rather poor and slow. There was marked nystagmus, coarse to the left, rapid to the right. Fundi: Right disk showed obscuration of the nasal margin with swelling of 2 D.; temporal margin fairly clear. There was marked tortuosity of the vessels, though they were not especially large. There were no hemorrhages or exudates. Left disk showed the same appearance, but here the swelling was only 1 D. Visual acuity (uncorrected): Right, 15/30; left, 15/50. Fields January 25 (Fig.

11) showed constriction of all boundaries with color-interlacing.

*Operation.*—January 27. Enucleation of left cerebellopontine tumor.

February 10. The fields showed widening of the color-boundaries but on the left colors remained somewhat interlaced. There was still some haziness of the nasal margins, though the swelling was less.

February 25. The fields (Fig. 12) showed restoration of normal color relations and widening of form fields. Some haziness of the disks persisted. Acuity: Right, 15/20; left, 15/40.

CASE 8.—Surgical No. 22,602. *Unlocalizable cerebral growth with choked disk and interlacing. Subtemporal decompression. Return of normal relations.*

*History.*—Mr. C. A. S., aged 32, was admitted June 30, 1908, complaining of persistent headache for two years with occasional projectile vomiting, loss of memory and change in disposition. He had had what he called "spells" with mental confusion and dim vision, but with no loss of consciousness.

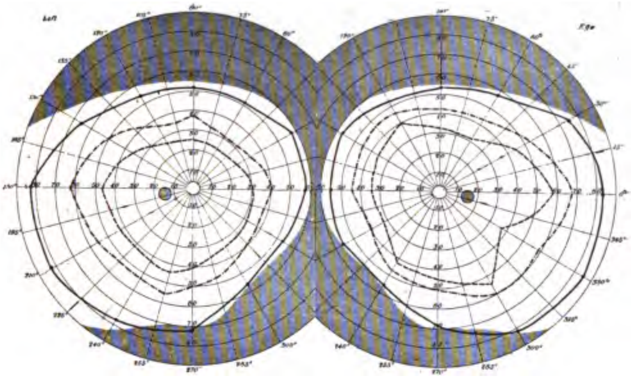

Fig. 14.—Charts of Case 8 thirty-eight days after operation, with normal color-relations and wide fields.

*Examination of Eyes.*—Pupils rather widely dilated; equal; react to light and on accommodation. Ocular movements normal. Fundi: Right disk swollen about 4 D.; marked edema of the nerve-head, extending well out into the retina. Retina had a peculiar watered-silk appearance. A single exudate on the disk; no hemorrhages. Arteries were somewhat small; veins very large and tortuous. Left disk also measured 4 D.; picture practically the same as in the right eye, except that there was one hemorrhagic area on the upper and outer side of the disk. Vision (subjectively obscured): Right, 20/20; left, 20/15.

Fields showed typical color-interlacing (Fig. 13) with but little constriction. Enlargement of blind spot.

*Operation.*—June 7. Left subtemporal decompression.

July 8. Both disks showed a number of small recent hemorrhages, but the swelling had receded to 3 D. and the retinal edema had subsided.

July 15. Subjective symptoms were entirely relieved; fields showed widening of the color boundaries with disappearance of interlacing (Fig. 14). Vision, 20/15 for each eye. Fundi showed swelling of 1 to 2 D. and some perivascular streaking; hemorrhages practically absorbed. The patient was discharged.

March 22, 1909. "It would be a problem to decide whether he has ever had choked disk" (Bordley).

The following case illustrates a further advance in Gunn's third stage, possibly approaching or actually in Stage 4; and yet beyond interlacing, which disappeared after the operation, and some persisting constriction, normal visual conditions were restored before the subsidence of the swelling:

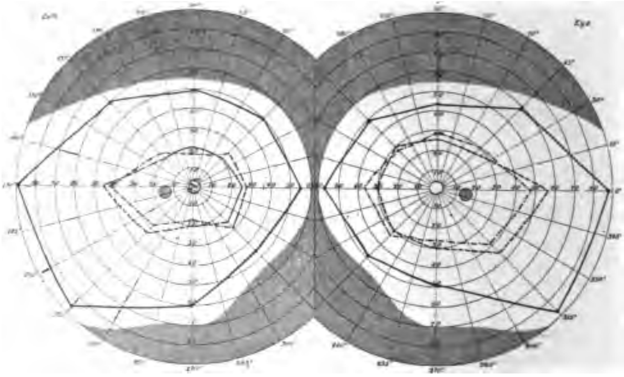

Fig. 15.—Charts of Case 9 before operation; interlacing with some constriction accompanying a condition of the papilla in an advanced third stage of choked disk.

Case 9.—Surgical No. 26,390. *Presumed cerebellar tumor with choked disk in advanced Stage 3 and interlacing. Postoperative restoration of normal color-relations.*

*History.*—Mr. G. W. H., aged 38, was admitted Aug. 12, 1910, giving a history of pressure-symptoms of a year's duration; at present severe suboccipital headaches, vomiting, ataxia, staggering gait, etc.; subjective blurring of vision for four months; diplopia for two months; attacks of temporary amaurosis frequent of late.

*Examination of Eyes.*—Pupils equal; reactions normal; palsy of right abducens; marked nystagmus, fine to right and coarse to left. Fundi: Right, swelling of 3 D.; disk fluffy and whitish; outlines completely obscured; cup filled and new-tissue formation apparent; veins tortuous; several hemorrhages undergoing absorption; Left, swelling 5 D. Appearance much the same as on right, but there were several more recent hemorrhages near the disk. Fields (Fig. 15) showed some constriction, especially on left, with interlacing. Acuity: Right, 15/30; left, 15/20.

*Operation.*—August 13. Suboccipital decompression. Rapid subsidence of general pressure-symptoms.

September 3. No diplopia; no observable weakness of right abducens; coarse nystagmus persisted. Fundi: Marked recession of edema, which was now barely measurable. Disks fluffy and opaque, with outlines still hazy; considerable tortuosity of vessels; hemorrhages practically absorbed; veins somewhat full and sinuous; arteries likewise; some evidence of new tissue in cup and along vessels. Fields August 29 (Fig. 16) showed resumption of normal color-relations, with some firm constriction.

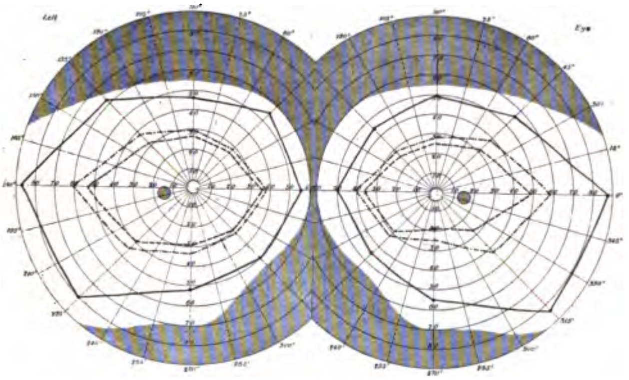

Fig. 16.—Charts of Case 9 sixteen days after operation, showing restoration of normal color-relations.

The following case (Case 10) is one well advanced in Gunn's fourth stage. The neuroretinal changes were not pronounced, but the perimetric deviations betray the severity of the process. There was marked constriction and interlacing, which was restored after operation about to the usual degree compatible with the advance of a choked disk to this stage.

**Case 10.**—Surgical No. 25,292. *Cerebral tumor, with choked disk in Stage 4. Interlacing and constriction, with post-operative restoration of color-relations.*

*History.*—Mrs. E. F., aged 40, was admitted Jan. 14, 1910, complaining of right-sided headaches for one year following trauma; nausea and vomiting of late; failing vision for eight months; transient periods of hemianesthesia, with some awkwardness in movement and paresthesias on the left; periods of temporary amblyopia during severe headaches.

*Examination of Eyes.*—Pupils equal and reactions normal; movements normal. Fundi: Swelling 3 D.; edges lost and cup much narrowed. One hemorrhage on right. No exudates; no excessive venous tortuosity. Fields (Fig. 17) showed constriction for form and colors with interlacing (1 cm. disks). Acuity: Right, 10/30; left, 20/30.

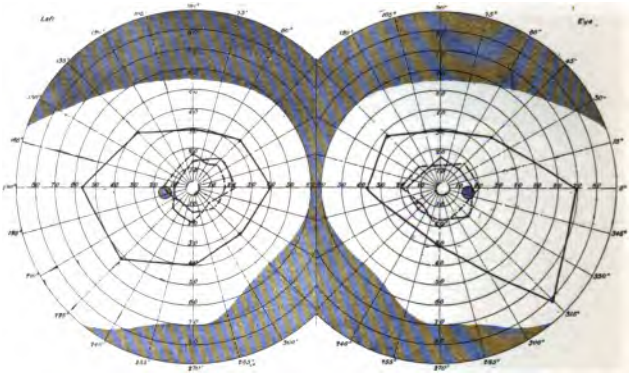

Fig. 17.—Cnarts of Case 10 before operation, plotted with 1 cm. disks, showing marked constriction and interlacing—Gunn's fourth stage.

*Operation.*—January 17. Combined exploration and decompression over right hemisphere revealed marked tension, but no surface lesion.

January 28. Fundi: Practically no measurable recession of edema. Fields showed restoration of normal color-relations, but remained constricted.

February 7. Fundi: Swelling had receded to 1.5 D. in each eye. Fields (Fig. 18) showed some widening, but otherwise remained as on January 28. Acuity lowered to right, 9/30; left, 15/30. Subjective condition much improved. Patient discharged.

June 16. The patient returned for observation, as protrusion had become somewhat tense of late. The patient's general condition was much improved; she had gained in weight, etc. Fundi still showed about 1 D. of swelling; disk margins

**obscured; perivascular streaks apparent. Acuity: Right, 15/40; left, 15/20.**

**September 27. The patient was readmitted, owing to appearance of localizing symptoms (astereognosis, etc.). Fundi showed an increase of swelling to 2 D.; new tissue formation abundant in cup and along vessels; distention of veins marked. Fields: Color-interlacing had reappeared on the left. The right field remained about as on February 7 (Fig. 19).**

**The patient subsequently developed a left homonymous hemianopsia, which is another story.**

Two cases have been selected to illustrate the perimetric deviations in Gunn's fifth stage. The first of these was a case of cerebral tumor, with the changes more advanced on the side corresponding with the involved hemisphere. In the second, a cerebellar case, the condition was about equal in the two eyes.

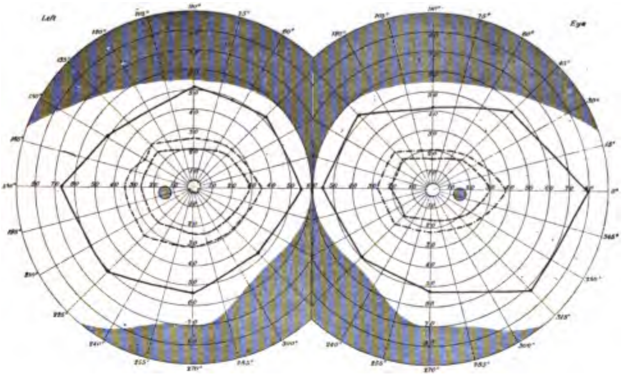

**Fig. 18.—Charts of Case 10 three weeks after operation, plotted with 1 cm. disks.**

CASE 11.—Surgical No. 25,090. *Cystic glioma of right frontal lobe. Choked disk in Stage 5, with marked impairment of vision on the right; Stage 4, with interlacing of color-fields on the left. Exploration with evacuation of cyst.*

*History.*—Mr. T. B. H., aged 28, was admitted April 18, 1910, with a history of neuralgic pains in the head for twelve years. several convulsive attacks with unconsciousness, paresthesias in left arm; headache during the previous year very severe; occasional vomiting. There had been some change of disposition and forgetfulness. Impairment of vision had been gradual. Frequent periods of amaurosis occurred, with occasional diplopia.

*Examination of Eyes.* — Slight exophthalmos on right. Pupils unequal; right 6 mm., left 5 mm. in moderate light.

Both reacted to light and distance. No oculomotor palsy. Fine nystagmoid twitches on looking up and to the right. Fundi: Right, swelling of 4 D.; marked distention and tortuosity of veins. Disk appeared opaque and glistening from definite atrophy; its outlines were recognizable though hazy. There were no hemorrhages, but numerous exudates. Left, swollen 5 D. Great tortuosity of vessels with distinct perivascular streaking. Evident atrophic changes. Fields (Fig. 19): Right, no colors recognized; constriction with tendency to nasal blindness; left, constriction and interlacing, with almost complete blue inversion. Visual Acuity: Right eye reduced to counting of fingers with difficulty; estimated acuity, 15/100; left eye, 15/30.

*Operation.*—April 23 and 27. Osteoplastic resection in two stages, with evacuation of large left precentral cyst was performed. Despite the marked general postoperative was performed. Despite the marked general postoperative improvement a further lowering of vision took place. On May 9 the fields (Fig. 20) showed a further constriction with a definite tendency toward a binasal defect. Normal color-relations, however, were restored. Fundi: Right, swelling of 1.5 to 2 D.; glistening appearance; perivascular streaking marked. Left, no measurable swelling; abundant new tissue formation, more evident than before. Acuity: Right, 3/100; left, 8/20.

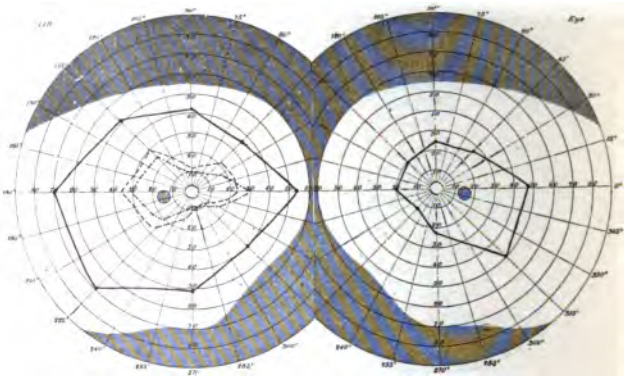

Fig. 19.—Charts of Case 11 before operation. On the left interlacing and constriction, with Stage 4 of choked disk; on the right, achromatopsia and marked nasal constriction in Gunn's fifth stage.

Aug. 12, 1910. The patient returned for observation, much improved. Fundi: Right, disk fluffy and glistening; no measurable swelling. Left had practically the same appearance; evidently a quiescent process. Fields practically the same as on May 9; the constricted color-field on the left

**showed no interlacing. Acuity: Right, reduced to counting fingers; left, 10/20.**

The history just recited shows that with adequate relief from the existing pressure disturbances a choked disk, even in this advanced stage, may subside and some vision still be preserved in spite of the contraction of the extensive new-tissue formation.

The following and last of the cases which we have selected to illustrate the terminal stage of Gunn's subdivisions was an acute case and, surgically speaking, a most unfavorable one. It illustrates, however, the existence of interlacing even to the final blotting out of central color perception. Though there was some lowering of vision after the decompressive operation, fairly useful sight (though not for reading) was preserved until the patient's death.

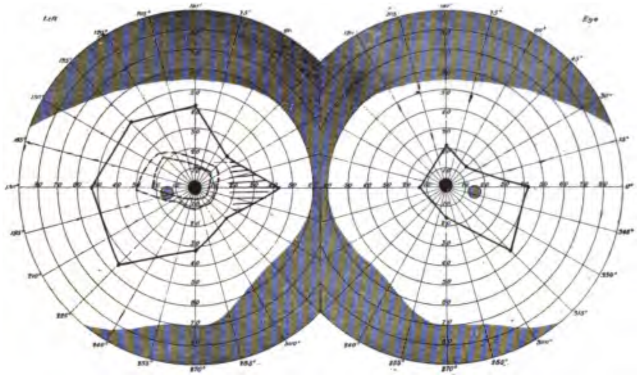

**Fig. 20.—Charts of Case 12 twelve days after operation. Restoration of color-relations on left, with advance in constriction for both colors and form.**

**Case 12.—Surgical No. 24,778. *Unlocalizable (presumably cerebellar) tumor. Choked disk in an advanced stage, with tendency toward a binasal defect. Bilateral decompression. Advancing condition halted short of blindness.***

***History.*—Mr. W. V. M., aged 40, was admitted Oct. 6, 1909, complaining of severe occipital headaches for eighteen months, occasional vomiting, blurring of vision for five months, attacks of total amaurosis for two months and at time of admission occurring with every change of position, with marked photophobia. Neurologic study suggested subtentorial lesion.**

***Examination of Eyes.*—Some slight exophthalmos; more marked on left than on right. Marked congestion of palpebral**

venules. Pupils wide, right slightly larger than left; reacted well together to light, distance and consensually; right less responsive alone. No loss of muscle balance. Some suggestive nystagmoid twitches. Fundi: Right, swelling of 9 D.; edema extended two disk diameters onto retina. The disk was very prominent; peaked at the center; the edge overhung the retina. Numerous yellowish exudates and white connective tissue deposits; no hemorrhages. The veins were full and excessively tortuous, dipping in and out of the edema. The arteries were small. Left eye showed practically the same condition; swelling of 7.5 D. Fields (Fig. 21): marked and irregular constriction, especially of color-fields, which interlaced. Acuity: Right 48/60; left, 20/60.

*Operation.*—October 8. Right subtemporal decompression was performed.

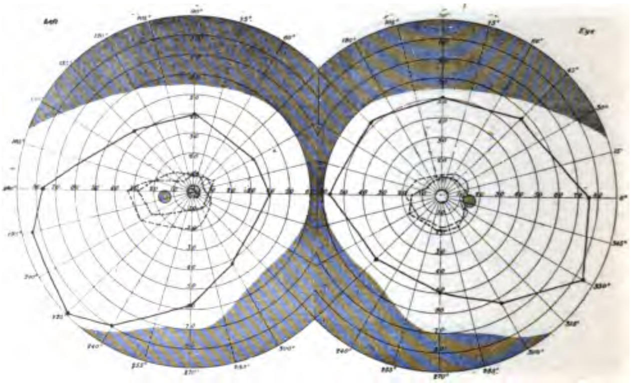

Fig. 21.—Charts of Case 12 before operation, taken with 1 cm. disks. Marked color constriction with interlacing in Gunn's fifth stage.

October 11. Fundi: Right, swelling 7.5 D.; left, 6.5 D. No change in general appearance.

October 14. Fundi: Right, 6.5 D.; left, 6.5 D. Subjective improvement in acuity. Cessation of attacks of amaurosis.

October 19. Fields had widened somewhat. Fundi: Right, 6 D.; left, 6 D. Retina less edematous. Acuity: Right, 30/60, left, 20/60.

October 21. Acuity: Right, 21/60; left, 12/60. This rapid lowering made further decompression seem urgent, and on October 23 a left subtemporal decompression was performed.

November 1. Fundi: Right, 5.5 D.; left, 5 D. No retinal edema. Acuity: Right, 21/60; left, 6/60.

November 8. Fundi: Right, 5 D.; left, 4.5 D. No change in general appearance, though veins were less tortuous. Fields

(Fig. 22) showed further constriction, with especial loss on nasal side, where colors were no longer perceived. Acuity: Right, 9/60; left, 3/60. Patient discharged.

November 24. The patient returned for observation. Fundi: Right disk measured 2 D.; disk margin coming into view; fluffy appearance from abundance of new tissue; obvious atrophy. Left disk less clear; 3 D. of swelling; margins still obscured. Acuity not so good as on November 8. Fields not taken. Patient was subjectively much improved, but the protrusions were tense and the suggestive cerebellar symptoms more pronounced. There was obviously an internal hydrocephalus. Suboccipital exploration was refused.

Vision continued about at this stage for two months and then improved somewhat. In March, 1910, he was able to write some and on "good days" could read large print, according to his wife's statement. He fell under the spell of Christian Science and died Oct. 15, 1910. No examination.

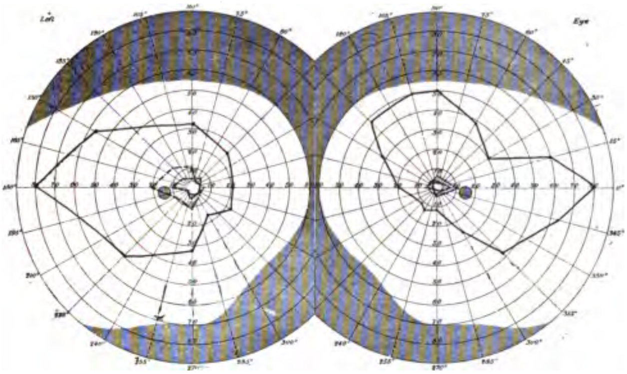

Fig. 22.—Charts of Case 12 a month after operation (1 cm. disks), showing further constriction, especially of nasal fields, with nasal hemiachromatopsia.

It is thus apparent that even in this final stage of Gunn's classification some vision may be preserved after operation and that even though the color-fields are greatly constricted they may still show interlacing of their boundaries. The terminal perimetric distortion for both eyes in this case exhibits a tendency toward a binasal defect, particularly for color-perception. A defect of this character was likewise apparent for the right eye in Case 11, the condition having progressed, however, to the stage of complete achromatopsia. These last two cases will be found to merge into the group of twelve cases, mentioned in our statistical analysis, which exhibited a final tendency toward a binasal hemianopsia with loss of color vision, a small central area of bitemporal vision for form alone persisting after the postoperative subsidence of the advanced grades of choked disk.

A full description of these twelve cases of so-called binasal hemianopsia will be reserved for another paper in this series. They are here referred to in order to show that the prognosis for some postoperative preservation of vision is less unfavorable in this fifth stage than Marcus Gunn was led to believe.

## SUMMARY

Out of the 123 cases in our tumor series in which perimetric observations could be made, fifty-three showed simple color-interlacing or inversion with more or less constriction of the field boundaries.

In ten of these fifty-three cases the dyschromatopsia either actually preceded any recognizable ophthalmoscopic change in the eye-grounds or accompanied most incipient stages of choked disk. These distortions of the color boundaries, therefore, promise to be of some service in the making of a more precocious diagnosis of an increase of intracranial tension than is commonly ventured on. Thus, in a number of cases of early tumor extirpation, color inversion and interlacing, in addition to attacks of Jacksonian epilepsy, were the only indications of the lesion.

The perimetric deviations, observed before and after operation, in the remaining forty-two cases of simple dyschromatopsia, we have attempted to correlate with the accompanying grades of choked disk subdivided into six groups according to Marcus Gunn's classification.

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